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GASTROMEGALY AND CHRONIC DUODENAL ILEUS IN CHILDREN

BY

REGINALD MILLER, M.D., F.R.C.P.,
Physician, Paddington Green Children's Hospital,

with

H. COURTNEY GAGE, L.R.C.P., M.R.C.S.,
Radiologist, St. Mary's Hospital, London.

This paper is concerned with a series of nine children who exhibited enlargement of the stomach with visible gastric peristalsis or other signs of obstruction high in the alimentary tract. The similarity between the cases is sufficient to suggest that they all belong to one group originating in some form of duodenal obstruction. Two of the nine cases were submitted to operation and were found to be examples of chronic duodenal ileus, and it is suggested that the others of the series owe their origin to the same cause.

Gastromegaly is a convenient term for the enlarged and hypertrophied stomach which is the most striking clinical feature of such cases as these. Such a condition implies obstruction to the evacuation of the stomach and may be due to various causes ; and as the site and nature of the obstruction, and even its very presence, may be difficult to determine, it is useful to have a group name which covers all such cases and emphasizes their one most obvious clinical abnormality. Were, for instance, the diagnosis of chronic duodenal ileus in the present cases disputed (and the subject is one of great difficulty), they would remain as a type of gastromegaly of obscure origin.

Duodenal ileus and arterio-mesenteric occlusion of the duodenum are the terms given to the condition in which the duodenum is compressed by the root of the mesentery and (usually) the superior mesenteric artery, as they lie over the duodeno-jejunal flexure. Both acute and chronic duodenal ileus are well recognized conditions in adults. This paper is concerned only with the chronic type, and as it occurs in children.

I. DUODENAL ILEUS IN ADULTS.

There is a considerable literature dealing with duodenal ileus in adults, and since the beginning of the present century the condition seems to have been almost universally recognized.

Credit is usually given to Rokitansky[1] as being the first to recognize the possibility of compression of the bowel by the root of the mesentery. This was in 1842, and in 1863[2] he described compression of the duodenum by the superior mesenteric artery as occurring in elderly people with visceroptosis. Heschl[3] (1855) and Glénard [4, 5] (1885 and 1889) were prominent writers on the subject. Clinically acute duodenal ileus, usually post-operative, first attracted

attention, and in 1891 Kundrat[6] reported three such cases. Others, mainly isolated examples, were reported in Germany and England. Presently it was noted that in some instances, in spite of the fact that the final symptoms were acute in onset and of short duration, hypertrophy of the stomach and duodenum was present; and this led to the description of chronic duodenal ileus. By 1899 Albrecht[7] was able to collect many cases and write a very full account of the condition, describing both acute and chronic types, and giving details of treatment by posture, lavage and gastro-enterostomy.

In France, according to Duval, chronic duodenal ileus was first described by Petit[8] in his thesis 'On the strangulation of the duodenum at the level of the mesenteric root' (1900). Many papers on the subject have appeared in the French literature, and special mention must be made of the chapter on the condition in the book 'The Duodenum' by Duval, Roux and Béclère[9].

In America duodenal ileus has also attracted much attention since 1900. Amongst those who early interested themselves in the subject must be mentioned Robinson[10, 11] (1900), Finney[12] (1906), Barker[13] (1906), Bloodgood[14] (1907), and Staveley[15] (1908). In 1911 and 1912 there appeared further communications by Staveley[16] and Bloodgood[17]. In 1921 the Kelloggs[18] reported a series of 41 cases all submitted to operation, and in 1926 Higgins[19] published 56 personal cases. The treatment of chronic duodenal ileus by duodeno-jejunostomy is said to have been first suggested by Barker[13] and Bloodgood[14], and the operation first performed by Staveley[15].

In England, although acute arterio-mesenteric ileus became a recognized entity, practically no attention was paid to chronic duodenal ileus until the publication by Wilkie[20] of his first paper on the subject in 1921, and this with his two subsequent communications [21, 22], still remain the chief contributions to the study of chronic duodenal ileus in the English literature.

The subject of chronic duodenal ileus in adults has excited much discussion, particularly over the two points of its frequency and pathogenesis.

As regards the first point, those who have specially interested themselves in the condition are inclined to lay stress upon its comparative frequency; but the general English surgical opinion at the present time seems to be that, although chronic duodenal ileus has won for itself full recognition, it remains a rarity, and that its frequency can easily be exaggerated even after inspection of the parts at operation.

The explanation of the mechanism by which obstructive symptoms are produced in chronic duodenal ileus forms rather a difficult and complicated problem. It is thought, in the first place, that there must be a congenital factor, an abnormal anatomical development, constricting the space through which the duodenum passes under the mesenteric pedicle and the superior mesenteric artery. The relative importance of this factor has been much debated. If it does not operate it is difficult to see why obstructive symptoms of duodenal ileus should arise in later life in some cases of visceroptosis and not in others: while, on the other hand, if the congenital factor is of real importance, it is, as several writers have argued, difficult to understand why the condition is so little known amongst children. A consideration of chronic duodenal ileus in children may be expected to throw an interesting light on this particular point, and it is gratifying to feel that the cases here recorded supply, as it were, a long-felt want.

It is thought that in adult cases this congenital anatomical factor at the most predisposes to the development of obstructive symptoms in later life, and is not of itself sufficient to cause these symptoms until additional and accessory factors come into play. These, by dragging on the mesentery

and so further reducing the space through which the duodenum passes, lead to actual compression of the bowel. These accessory factors concern the stomach, small intestine and colon. Visceroptosis is regarded as the commonest of them, and this accounts for the age incidence of chronic duodenal ileus and its greater frequency in women than in men. Distension, overloading and sagging of the proximal colon are the causes in another group of cases. Here the colon, by congenital formation, may be unduly mobile, and the artery traversing the duodenum may be the right colic and not the superior mesenteric artery. Glénard[5], Barton[23] and others regard ptosis, atony and dilatation of the stomach as able to lead to compression of the duodenum in a third group of cases. Distension of the stomach by air-swallowing has been put forward by Leveuf[24] as a possible factor. On this point Wilkie[20] expresses the view that air-swallowing may aggravate the condition but is not of primary ætiological significance.

In another group of cases which are rather different from the foregoing, in that no congenital factor need be postulated, disease of the mesentery or its glands produces direct compression of the duodenum. Tuberculosis and malignant disease have been reported as the agents responsible for such cases by Grégoire[25] and Wilkie[22].

Acute arterio-mesenteric ileus accounts for at least some of the cases described as 'acute dilatation of the stomach,' 'gastrorrhœa' and 'acute paralytic distension of the stomach,' which occur most commonly after laparotomy. As has been already mentioned, in some of the examples of this acute condition, hypertrophy of the stomach and duodenum has been recognized, and it is therefore supposed that many of them owe their origin to chronic duodenal ileus which has either been symptomless or undiagnosed. Wilkie[20] has expressed the opinion that 'acute dilatation of the stomach, either idio- or post-operative, is probably a gross manifestation of a previously present chronic condition.'

II. DUODENAL ILEUS IN CHILDREN.

The amount of attention which has been paid to duodenal ileus in children is scanty, and there is no paper in the literature devoted to a consideration of the subject. On the other hand there are some isolated references to it, and an attempt is made here to summarize these so as to give some idea of the present position of affairs with regard to this condition. No cases altogether similar to the series reported here seem to have been published hitherto.

Acute ileus.—Acute duodenal ileus of the type seen in adults does not seem to occur in children. There is no mention of it in the literature, and surgeons of great experience in pædiatrics do not seem to have met it. Although this paper is concerned only with the chronic type of duodenal ileus, it is proper to mention the non-occurrence of the acute cases. As the acute type is thought to be predisposed to by preceding chronic compression, its absence in children might be taken as evidence against the occurrence of chronic cases in them. But the evidence in favour of their occurrence is far stronger, and probably the absence of the acute cases is due to the fact that severe atony and ptosis of the obstructed stomach are unlikely to occur in children.

Chronic ileus.—Chronic duodenal ileus in children has been described in isolated examples; but before passing to these and the groups into which they fall, there are two points bearing on the question which must be mentioned.

One has already received attention, namely, the question raised in discussing the pathogenesis of chronic duodenal ileus, why so little is known of the condition in children. This point has been pressed, for example, by Gracie[26], and the significance of the argument has been explained earlier in this paper.

The second point is of greater interest and consists in the fact that a proportion of cases operated on for chronic duodenal ileus in adult life give histories of attacks of vomiting in early life. This observation, which clearly has an important bearing on the present subject, was made by Wilkie[22] in 1927 and has recently been confirmed by Hayes and Shaw[27]. On this point Wilkie[22] has written:—

> Not infrequently these attacks (of bilious vomiting) date back to early childhood and are regarded as constitutional or as evidence of recurring acidosis. Such attacks may disappear during late adolescence and the patient may enjoy comfort for ten or fifteen years, when there appear the flatulent symptoms which bring the patient to seek relief. This late reappearance of symptoms may follow child-bearing, some illness, such as influenza, or periods of mental worry associated with deficient muscular exercise and fresh air, in fact with any condition which tends to lower the tone of the abdominal muscles and to favour visceroptosis.

No detailed description of these cases in their childhood has been given. In view of this observation it is of interest that the series of cases to be reported in this paper has shown instances in which spontaneous improvement was taking place. In this respect they differ from any other cases of chronic duodenal ileus in children as yet reported, and it is difficult not to conclude that the present cases are of the type referred to by Wilkie, but diagnosed during childhood. If this assumption is correct, an important matter in ultimate prognosis is involved.

Passing to the published cases of chronic duodenal ileus in children, these are found to fall into two main groups, neither of which is altogether similar to the present series of cases. It is sufficient here to give a general description of the types of these recorded cases: synopses of the case reports will be found in Appendix B.

Group 1. Cases with megaduodenum. This group consists of rare cases, usually in very early life, in which the arterio-mesenteric compression is severe enough to produce great enlargement of the duodenum. In them the duodenum is found as a visible and palpable tumour, and may even show peristalsis. In some instances it is described as the size of a second stomach, and skiagrams easily demonstrate the enlarged duodenum extending beyond the enlarged stomach. The symptoms are severe, and consist of vomiting and its consequences. Such cases are to some extent comparable clinically with hypertrophic pyloric stenosis, from which they are distinguished by the

absence of a pyloric tumour, the presence of bile in the vomitus, and the signs of megaduodenum. They may also be simulated by congenital duodenal stenosis. The presence of some form of obstruction is apparently obvious from the severity of the vomiting. Some cases have been successfully treated by duodeno-jejunostomy. They show no tendency towards spontaneous improvement.

The first of these cases was reported by Bernheim-Karrer[28] in 1904, and since then further examples have been published by Frank[29], Downes[30], DuBose[31], Jewesbury[32], and Henske and Best[33] (see Table 1 and Appendix B). Megaduodenum may be due to other causes than arterio-mesenteric compression. Many cases have been recorded as due to obstruction by peritoneal bands, a class of case recently reviewed by Higgins and Paterson[34]. It has also been recorded as due to congenital duodenal stenosis by Jewesbury[32], and by Variot and Cailliau[35]; and to pressure from a tumour on the duodenum in a new born infant by Hohlbaum[36].

TABLE 1.

RECORDED CASES OF MEGADUODENUM DUE TO ARTERIO-MESENTERIC COMPRESSION.

Author.	Date.	Sex.	Age of onset of symptoms.	Age when first seen.	Enlargement of duodenum.				Result.
					Clinical.	X-ray.	Autopsy.	Operation.	
Bernheim-Karrer[28]	1904	M	Birth	8 months			Greatly dilated		Died at 8 months
Frank[29]	1913	F	4½ months	11 months	Visible enlargement			Much enlarged	Duod.-jejunostomy: successful.
Frank[29]	1913	M	12 months	2 years	Visible enlargement	Enlarged			Operation recommended at 2½ yr., but refused.
Downes[30]	1917	M	Birth	4½ years	Visible enlargement & peristalsis			Enlarged	Gastro-enterostomy & division of pylorus: successful.
Dubose[31]	1919	F	3 days	2 months		Enlarged		Enlarged	Gastro-enterostomy & occlusion of pylorus: successful.
Jewesbury[32]	1922		3 days	9 days			Greatly dilated		Died.
Henske and Best[33]	1928		Birth	5 months		Dilated	Obstructed		Died at 21 mth.

The cases in this group are so much more severe in their symptoms and obvious in their physical signs that they bear no great resemblance to the present writer's cases: but their theoretical interest is considerable. They appear to prove beyond doubt that the congenital anatomical abnormality can be a very real factor in the production of arterio-mesenteric compression of the duodenum, and may of itself be sufficient to produce severe symptoms, even death, in early life. It is true that there has been some discussion (Duval[37]) whether megaduodenum might not be considered as an essential dilatation comparable to the megacolon of Hirschsprung's disease; but the evidence in favour of a congenital arterio-mesenteric compression of the duodenum is decisive.

GROUP 2. CASES OF ADULT TYPE. There are a few cases reported of the occurrence in children of the adult type of chronic duodenal ileus. The details given of these cases are unfortunately very scanty, but the few examples which can be quoted seem to form a group in which the congenital compression of the duodenum is not itself sufficient to give rise to symptoms. Thus the obstructive symptoms arise at a later age than in the first group, and are probably due to the action of accessory factors, as in adults. Only one case is reported with sufficient detail (Quain[38]) to be sure of this, but in this instance it seems clear that the onset of gastroptosis determined the onset of the symptoms. In this group the symptoms are of the adult type; that is to say, painful dyspepsia of obscure origin, flatulence and attacks of vomiting.

The first case of this type was reported by Quervain[39] in the first edition (1907) of his book on 'Clinical Surgical Diagnosis' (page 273), and it is still contained in the current English edition of the book. Quain's[38] (1920) case was in a girl of 17 years who started to have symptoms at the age of 8 years. Higgins[19] (1926) recorded a case in a boy of 7 years who had suffered from epigastric pain and attacks of vomiting 'for some months.' Wilkie[26] (1927) has stated that the most marked case of duodenal ileus which he has met was in a boy aged 7 years. Hayes and Shaw[27] (1929) refer without details to such a case (Case 21). Such details of these cases as are available are given in Appendix B.

These two groups contain all the straightforward cases of chronic duodenal ileus in children ætiologically comparable to the ordinary cases in adults. It will be seen that, even allowing a margin for references that cannot be traced, the number of the cases in children is very small. There are, however, certain other cases showing differences from the foregoing which can be collected into two subsidiary groups.

GROUP 3. CASES WITH ASSOCIATED CONDITIONS. As might be expected to be the case in children, chronic duodenal ileus has been found associated with other conditions and mal-developments. Cases showing excessive mobility of the proximal colon, as in the ordinary colonic type of chronic duodenal ileus of adults are not included here.

Ombrédanne[40] (1919) has described a case in a girl aged 12 years complicated by volvulus. Wheelon[41] (1921) has recorded a case which should perhaps be included here. Foucar[42] (1923) has published two very interesting cases, the first of which falls into this group. A boy of 4 years showed visible gastric peristalsis, and when submitted to X-ray examination during an attack of vomiting, but not during a period of quiescence, showed duodenal obstruction. At operation adhesive fœtal peritonitis was found resulting in a volvulus of the small intestine,

which caused secondary and only intermittent closure of the lumen of the duodenum. Waugh[43] (1928) has reported the case of a boy aged 7½ years in which there was a tight constriction of the second part of the duodenum caused by a cystico-colic membrane, and also a tendency to arterio-mesenteric constriction. Case 2 of the present series is the converse of Waugh's case: here peritoneal thickenings were present but did not seem to be causing obstruction. Judd and White[43] (1929) have described two cases of duodenal compression in children, one with an abnormally placed mesentery and the other with mal-position of the duodenum. Higgins[19] (1926), who reported a series of 56 cases of chronic duodenal ileus, including one example in a child as already mentioned, has stated that this condition 'is of rather infrequent occurrence in children unless it is associated with some congenital abnormality.' Synopses of the above cases are given in Appendix B.

Group 4. Cases with disease of the mesentery. Rarely disease of the mesentery can produce compression of the duodenum in a child, as is shown by the following interesting case which clinically resembles the present cases more closely than any others previously recorded.

Foucar[42] (1923) has reported (Case 2) a case in a boy of 18 months. The child had suffered since birth from attacks of projectile, bilious vomiting, which recurred about once a week, and clearly demonstrated the presence of gastric stasis. Visible gastric peristalsis had been observed by the parents since the 10th month. X-ray examination, made during a quiescent period, revealed no obstruction. At operation the mesentery was found to be very œdematous owing to the presence of markedly enlarged glands.

In the presence of visible peristalsis and the absence of radiological evidence of obstruction, this case resembles those of the present series, exhibiting a latent rather than a chronic obstruction; and it is noteworthy that Foucar entitled his paper 'Intermittent duodenal obstruction in children' (see Appendix B.)

Summary.—Having reviewed the literature on duodenal ileus in children it will be well to summarize the present position of the subject. Acute cases of the adult type do not seem to occur in children. Chronic duodenal ileus in children has excited no little theoretical interest chiefly on account of the support which such cases would give to the presence of a congenital ætiological factor in the adult cases; and wonder has been expressed that more is not seen of the condition in children.

Only very few cases have been reported in children. In the commonest type the duodenal obstruction is so severe as to produce, usually during infancy, a megaduodenum, forming a visible and palpable tumour; and this group shows clearly that it is possible for the congenital factor alone to be responsible for very severe duodenal compression. Secondly, a very few examples of the adult type of chronic duodenal ileus have been reported in older children. There are also reports of ileus associated with other conditions and mal-developments, and one case of duodenal compression from disease of the mesentery and its glands.

No cases of true duodenal ileus in children have been found which resemble the present series of cases. The nearest reproduction of their signs and symptoms has been found in a single case of compression of the duodenum by enlarged mesenteric glands (Foucar[42], Case 2), which the author regarded as due to 'intermittent' obstruction of the duodenum.

III. PRESENT SERIES OF CASES.

The present series consists of nine cases of gastromegaly apparently due to mild duodenal obstruction. They appear sufficiently alike to warrant their inclusion in one clinical group. Two cases were proved at operation to be examples of chronic arterio-mesenteric compression of the duodenum, and the view is taken that the others are due to the same condition.

Characteristics of the series. Before considering the cases in detail it is well to give their characteristics as a group.

1. These cases do not conform to either of the two main types of chronic duodenal ileus which have been already described and reviewed in this paper. They are not so urgently severe as the infantile group with megaduodenum, nor do they show the same symptoms and course as the adult type which has been rarely reported in older children. Rather they seem to conform to the type whose existence has been foreshadowed by Wilkie[22] in his observation that a proportion of his adult cases gave a history of vomiting attacks in early life, succeeded in adolescence by a quiescent period lasting for many years.

2. The symptoms start in very early life and often from birth. They are for the most part mild. They vary in degree of severity in the different cases, but in no instance are they urgent or spectacular, and they do not readily lead to a suspicion of the presence of organic obstruction. The reason for this is that the congenital obstruction is only severe enough to set up gastric stasis and secondary catarrh (as in the pyloric stenosis of infants), and so loss of appetite is a more troublesome early symptom than is vomiting. The loss of appetite is so severe as to lead to limitation of the amount of food taken, and this limitation keeps the vomiting in abeyance. If this sequence of events is grasped it will be understood how the presence of organic obstruction is at first masked. Later, as the child gets on to solid food and can be pressed to eat against its will, vomiting becomes more troublesome, tending to occur in attacks, accompanied by toxæmia and diarrhœa.

3. Similarly, the physical signs of obstruction are not obtrusive and may require prolonged observation before they are recognized. Chief of them are enlargement of the stomach lying across the upper abdomen, and visible gastric peristalsis. In the shape and position of the stomach, and in the care which has to be expended to elicit visible gastric peristalsis, these cases are rather reminiscent of examples of hypertrophic pyloric stenosis.

4. X-ray examination confirms the presence of obstruction to the evacuation of the stomach, and excludes the stomach itself as the site of the obstruction. It does not show, at all events during periods of freedom from vomiting, the presence of obstruction in the duodenum.

5. The cases as a whole show a tendency towards gradual recovery. This is presumably due in some way to a lessening of the constriction at the duodeno-jejunal flexure by the growth of the child.

6. The obstruction is at first persistent with intermittent exacerbations due usually to gastric distension: but as spontaneous improvement sets in,

it is probable that only the intermittent obstructive factor is at work, and at this stage suitable dietetic treatment may lead to the cessation of symptoms (see Cases 3 and 7).

Pathogenesis.—In Cases 1 to 7 inclusive it is clear that if the diagnosis of chronic duodenal ileus is correct, compression of the duodenum was present at birth, and that it was the congenital anatomical factor which was responsible for the early symptoms of anorexia, limitation of food, vomiting and malnutrition in so far as these symptoms were present. In Case 8 the history of the first five years of life was unobtainable; and in Case 9 there was so much distension of the colon that it is impossible to assess the importance of any congenital factor in the production of the duodenal compression.

Turning to the influence of the accessory factors, as seen in adults, in producing obstructive symptoms, there seem to be differences to note in these children. In Case 2 the hypertrophy of the gastric wall had 'failed,' and some degree of gastroptosis was present. In Case 9 the abnormality of the colon was sufficient to suggest that this was an example of the colonic type of duodenal ileus as described in adults. In Case 8 the colon was also rather voluminous. But in the other cases it seems impossible to incriminate any of the ordinary accessory factors. Yet distension of the stomach seems undoubtedly to increase the obstruction in these cases, and in the absence of gastroptosis some other explanation is required. When it is remembered how far the distended gastromegalic stomach passes over to the right, the pyloric portion of the stomach coming to lie right over the pylorus, it may be suggested that this displacement adds to the obstruction at the root of the mesentery.

Frequency.—Two of the nine cases were seen some years ago (Case 9 in 1923, and Case 7 in 1926). In Case 9 the presence of obstruction to the evacuation of the stomach was not recognized until 1929; and Case 7 which showed marked visible gastric peristalsis when first seen, was not understood. Case 1 came under the writer's care in December, 1928, and the exact diagnosis was determined at operation in May, 1929. In the twelve months from December, 1928, therefore, seven cases were seen. During the same period three other cases were met. One was the man referred to in the section on prognosis, apparently a recovered case. The second, a most marked case of gastromegaly, was a boy seen in a convalescent home by the courtesy of Dr. Hugh Raven, and not included in the series owing to lack of clinical details available. The third was a boy seen at hospital in whom the evidence of obstruction was insufficient for publication.*

It is, of course, a commonplace that cases of special interest are always met in droves, yet the experience of the past year is suggestive. In four out of the nine cases visible gastric peristalsis was proceeding at the time of their first examination, though it had not been previously noted: in the other five cases a good deal of time and trouble was spent on proving the existence of obstruction. It is therefore possible that were evidence of obstruction looked for in older children with the care that is expended on the same point in young babies, cases of gastromegaly due to chronic duodenal ileus of the rather latent type described here would be found of no great rarity.

* Another case (female) was shown by me at the Children's Section of the Royal Society of Medicine on Feb. 28th, 1930.—R.M.

Sex incidence.—Of the nine cases of the series only two were in girls. The three additional examples mentioned above were all males. No great stress can be laid on the sex incidence worked out on only (at the most) twelve cases. But the point needs mention here because the sex incidence of chronic duodenal ileus in adults shows a preponderance of females over males. This is attributed to the greater frequency of visceroptosis in the female sex. In the present series in children the factor of visceroptosis is of little importance, the congenital factor being of chief moment. If, then, the sex incidence as mentioned above is more than accidental, it may be that it reflects the greater frequency of congenital anatomical abnormalities in the male sex.

Synopsis of present cases.

It is convenient to give here only the salient clinical features of the nine cases included in the present series. The full case-records are given in Appendix A.

Case 1. B.R., male, born February, 1926. At birth he weighed 5¼ lb. and was frail and atonic. During the first year his appetite was so bad that feeding was very difficult. During the second year vomiting began and became worse after the 18th month. It occurred in attacks usually following attempted increases in the diet. Preceded by hiccup, extra coating of the tongue and refusal of food, vomiting usually started at night. The vomit was forcible, copious, containing food and bile-stained mucus. Diarrhœa with much mucus in the stools immediately followed the vomiting. Progress during the second year was very slow. He was first seen by the writer in July, 1928, aged 2 years and 4 months. He was very thin (18½ lb.) but not toxic in appearance; nor was he stunted in growth except by the presence of scoliosis. Gastric peristalsis was observed at this date for the first time. An X-ray examination showed enlargement of the stomach but no gastric stasis.*

The boy came under the care of the present writer in December, 1928. On a dry diet it was possible to avoid the attacks of vomiting, but not to give enough food to lead to progress. An increase in the amount of food given would in one or two days lead to hiccup, increased gastritis, increased refusal of food, visible gastric peristalsis: obvious preludes to an attack of vomiting. In February, 1929, a second X-ray examination was made (H.C.G.). This showed a large stomach with good peristalsis but considerable stasis. The duodenal cap appeared normal and the distal parts of the duodenum were not seen (Fig. 1). The abdomen was opened in May, 1929, and there was found chronic duodenal ileus with enlargement and hypertrophy of the stomach and duodenum, and atrophy of the jejunum. A posterior gastro-jejunostomy was performed. After operation, although the symptoms of obstruction were relieved, the stomach decreased in size, and the chronic gastritis cured, only slow progress was made. In February, 1930, a third X-ray examination (H.C.G.) showed that the stomach retained an 8 oz. meal for two or three hours, and was empty in 3 hours. This result was so satisfactory that the comparative lack of progress since the operation appeared due in part to the difficult psychology of the child and in part to defective tissue assimilation.

Case 2. A.E., male; first seen October 3rd., 1929, aged 5 years. Was brought up for vomiting and wasting. At birth he weighed 8¾ lb. From birth he took feeds slowly and without appetite. At 7th week began projectile vomiting and was suspected of pyloric stenosis, but was ultimately diagnosed as 'indigestion.' Vomiting continued off and on, and at 18 months on solid food it increased. It recurred every 3 or 4 weeks in attacks accompanied by fever, drowsiness, rapid loss of weight with mucus in the stools. Hiccup was a frequent symptom. He continued on these lines, eating extraordinarily little, so that at 5 his condition was worse than at 3 years.

At 5 years he weighed 34 lb. 14 oz. He was quite an invalid child and his appetite was extremely bad, amounting to refusal of food. On the very meagre diet he could be prevailed upon to take, there was no vomiting. The abdomen showed enlarged stomach, gastric splashing

* The X-ray examinations for which Dr. Gage is responsible are initialled (H.C.G.). Every photograph has been reduced on the same scale, so that all are strictly comparable to each other.

and feeble visible peristalsis. The wall of the stomach felt thickened. The stools were fatty. X-ray examination (H.C.G.) showed a large atonic stomach, very poor peristalsis and severe gastric delay (Fig. 2).

A diagnosis was made of duodenal obstruction with failure of the compensatory gastric hypertrophy. At operation (November 11th., 1929) chronic duodenal ileus was found.

The stomach was found enlarged, flaccid, but showing hypertrophy. The duodenum was enlarged and hypertrophied so that it appeared the size of an adult duodenum. This change was apparent as far as the duodenum could be traced (first and second parts), and the jejunum was normal. No disease of the mesentery or its glands was found, but a small amount of unexplained clear fluid was present in the abdominal cavity. A posterior no-loop gastro-jejunostomy was performed.

In the ensuing three months very slight progress was made, both the lack of tone of the stomach and the psychological condition of the child proving severe handicaps to progress.

Case 3. M.M., girl, aged 5½ years. Birth weight 4 lb. From birth there were great difficulties in feeding and fairly frequent vomiting of projectile type. With the vomiting occurred diarrhœa with mucus and sometimes blood in the stools. The baby was habitually cross. Hiccup was common. Feeding seemed to give rise to discomfort. Refusal of food continued throughout the 2nd, 3rd and 4th years, and progress was very slow. The vomiting settled down into attacks typical of gastric stasis : the diarrhœa continued to be associated with the vomiting. An X-ray examination at the age of 5 years and 5 months showed a large stomach covering over the pyloric region. Emptying time was only 2 hours.

First seen at 5½ years (December, 1928). Weight 36½ lb. Since the X-ray examination she had been put on a dry diet and no vomiting had occurred. The stomach was large, its walls felt hypertrophied, and splashing was elicited. No gastric peristalsis was seen at that time. The dry diet was continued.

In March, 1929, she was seen again. She had continued free of all vomiting and nausea and had gained weight. An X-ray examination (H.C.G.) showed the stomach was average in size, its peristalsis suggested hypertrophy, but there was no stasis. The duodenal cap appeared normal (Fig. 3).

In September, 1929, definite visible gastric peristalsis was seen after inflation of the stomach. There had been no return of the vomiting.

Diagnosis. Although proof of obstruction to the emptying of the stomach was in this case difficult to obtain, the appearance of visible gastric peristalsis ultimately confirmed what the history of the case and the thickening of the gastric wall had at first suggested. In view of the similarity to Case 1 a diagnosis was made of chronic duodenal ileus in which symptoms were passing off.

Progress was satisfactory on the régime of a dry diet with gastric antiseptics. No vomiting occurred for 16 months, and the appetite improved.

Case 4. H.D., male, aged 4 years. Brought (November 28, 1929) for malnutrition and diarrhœa. Always frail and had made slow progress. For the past year no progress had been made. Was subject to attacks of diarrhœa, with large, pale and offensive motions. Hiccup was frequent. On very restricted diet owing to the diarrhœic attacks, he eat well and was not sick. At 4 years he looked very frail ; weight, 28 lb. His tongue was coated, and breath foul. The upper abdomen was large and the outline of the enlarged stomach was clearly seen. Its wall felt thickened and gastric peristalsis was observed. X-ray examination (H.C.G.) showed an enlarged stomach tending to hide the outlet. Peristalsis was powerful and suggested hyper-peristalsis. There was a small residue at 3½ hours. The duodenal cap was normal (Fig. 4).

Diagnosis : duodenal obstruction, probably due to chronic duodenal ileus.

Progress on pounded food with drinks in between meals was satisfactory and resulted in gain of weight.

Case 5. D.M., male, aged 2 years and 4 months. Brought (May 29, 1929) for enlarged abdomen, which had been present since the age of 9 months. There were no symptoms of ill-health. His weight was 25 lb. The upper abdomen was very protuberant. The stomach outline was clearly seen, vigorous gastric peristalsis was visible, and the wall of the stomach felt definitely thickened. Splashing was easily elicited. The tongue was slightly coated.

The bowels normal. The X-ray examination (H.C.G.) showed that the stomach was much enlarged. Peristalsis was vigorous and indicative of hypertrophy. More than 50% of the meal remained in the stomach at 3½ hours. The duodenal cap appeared normal. The colon was distended and perhaps large (Fig. 5).

Diagnosis: duodenal obstruction probably due to ileus.

As there were no symptoms and good progress was being made, no treatment was ordered.

Case 6. A.A., male, aged 6 years. Brought (November 15, 1929), for asthma. Appetite had always been rather poor, and became worse with change on to solid food at 18 months. Recently appetite had been very poor and there were periods of refusal of food. For past 2 years his weight had gone down. There had been only occasional vomiting, but when it occurred it was projectile in type. There was much flatulence and persistent hiccup after meals. Much gurgling in the stomach had been heard. The upper abdomen was protuberant. The outline of the stomach was clearly visible. Well marked peristalsis was seen. The wall of the stomach felt thickened. X-ray examination (H.C.G.) showed the stomach apparently only a little larger than normal. Hyper-peristalsis was present at first but died down later. There was a large residue at 4¾ hours. No view of the duodenal cap was obtained (Fig. 6).

Diagnosis: mild obstruction probably due to chronic duodenal ileus.

Pounded and minced foods were ordered to be taken as dry as possible. Drinks only in between meals. The foot of the bed was raised at night. With these measures the stomach became less obvious and the peristalsis much less in evidence.

Case 7. (First case seen.) E.McC., male, aged 8 years. Brought up in January, 1926, for poor digestion. Had always been a very poor feeder. No pain and seldom sick. Hiccup was very persistent. Abdomen was said to have been enlarged from 18 months to 6 years. Recently he had shown improvement. Bowels rather constipated: attacks of diarrhœa occurring about every two months. At 8 he was 49 in. in height, pale and thin. The upper abdomen was enlarged, the outline of the stomach clearly visible, its walls felt thickened, and well marked peristalsis was seen. X-ray examination showed a very large stomach hiding the duodenal cap and emptying slowly (Fig. 7).

It was recognized in 1926 that there was obstruction to the outlet of the stomach which did not show in the skiagrams. It was thought that this might be due to kinking in an enlarged atonic stomach. It is now suggested that the case was one of chronic duodenal ileus. On dry meals improvement occurred. At 11 he weighed 66½ lb., and was at school eating ordinary diet. His appendix had been removed without any post-operative complications.

Case 8. J.G., male, aged 12 years. Brought (July 31st, 1929), for mal-shaped abdomen. Early history not obtainable. Abdomen was enlarged by age of 5 years. Has progressed slowly and is subject to attacks of vomiting occurring about four times a year. Vomitus contains bile. Is at boarding-school, and is showing general improvement especially since he started drilling exercises. Tonsils were removed at 10 without incident. The upper abdomen was very large where the stomach could be seen much distended. Splashing was obtained but no peristalsis could be seen even after inflation of the stomach. X-ray examination (H.C.G.) showed enlargement of the stomach with normal peristalsis and emptying time. Duodenal cap was normal. No ileal stasis was observed, but the colon was voluminous and the transverse portion dipped low in the pelvis in the erect position. There was considerable colonic stasis (Fig. 8).

The diagnosis of duodenal obstruction is not clear, yet the emptying of such a large stomach in normal time suggests gastric hypertrophy. It is suggested that this is a case of chronic duodenal ileus in which the voluminous colon acts as an accessory factor in producing the symptoms of obstruction. No such symptoms were present when the boy was under observation.

Case 9. D.N., girl, first seen at the age of 2 years and 4 months (September 1925), for enlarged abdomen. The abdominal enlargement had been present for at least a year. There were no gastric symptoms except flatulence. She eat hungrily of the restricted diet permitted. She was not thin and had a good colour. She was subject to alternate attacks of diarrhœa and constipation. X-ray examination showed great dilatation of the stomach and colon (Fig 9). The stool contained a mild excess of fat. She was kept on a fat-free diet which seemed to keep her free of flatulence. In March, 1928, a second X-ray examination showed much the same condition as before. In May, 1929, in distending the stomach with a draught of soda water

well-marked gastric peristalsis was observed. It is suggested that this is a case of chronic duodenal ileus in which the 'drag' of the colon is the chief factor in producing obstruction of the duodenum. In 2¾ years she had gained 16¼ lb. and grown 7½ inches. Treatment on the same lines is being continued.

Symptomatology.

It is noticeable that the severity of the symptoms varies very much in the nine cases recorded here. In Cases 1, 2, 3, and 4 the degree of invalidity was considerable, and the children had been under medical supervision most of their lives. On the other hand, in Case 5, although the signs of obstruction were very definite, there was almost complete freedom from symptoms except for enlargement of the abdomen. In the other cases the degree of illness occasioned by the condition lay between these two extremes.

The mechanical factors at work in the production of symptoms have already been discussed, and presumably the variation in the severity of the symptoms depends chiefly on the variation in the degree of obstruction with which the child is born, and whether spontaneous improvement has set in. In addition to these mechanical factors, there are two other factors which may have a minor influence on the production of the symptoms, namely, the capacity of the gastric wall to undergo compensatory hypertrophy, and the chronic gastritis which may accompany the gastric stasis. The first must depend on the condition of the child at birth, its capacity to progress, and its freedom from debilitating illnesses, and so on.

Date of onset of symptoms.—As might be expected it is impossible to give any precise date at which symptoms become recognizable as indicating organic obstruction. In the worst instances difficulties arose from the earliest days. In Cases 1 and 3, from birth there was great difficulty in getting the child to take its feeds, although neither was regarded as an ill infant at first. Cases 2 and 7 had been taken as infants to specialists on account of indigestion and failure to progress. Case 4 at nine months was regarded as a delicate child but was thought to be free of anything that would ultimately prove harmful.

Where symptoms could be traced back to the earliest days it was found that they appeared worse in the second than in the first year of life, and in several instances they definitely increased in severity when the child began to take more solid food at about the age of eighteen months. In more than one case the mother has stated that whatever might be wrong with the child's stomach, it was certainly present from birth; and this statement may be taken to prove that there is no particular time at which there is a development of symptoms in the sense that a previously healthy child becomes an ill child.

The ages of the nine cases when they were first seen by the writer are not without interest for the reason that in no instance had the presence of any obstructive factor in the case been previously recognized. These figures, together with the dates of the onset of symptoms are given in Table 2.

Earliest symptoms.—Emphasis has already been laid on the fact that the earliest symptom may be loss of appetite rather than vomiting. The limitation of the amount of food which the child can be made to take keeps the vomiting in abeyance at first. Malnutrition and lack of progress are present in most cases.

Diarrhœa, especially associated with the attacks of vomiting, may be an early symptom. Taken as a whole the early symptoms have two points of interest about them : their nature depends largely on the amount of food which the child is given, and is more suggestive of indigestion than of obstruction.

Symptoms.—The symptoms may now be considered individually.

Anorexia. In the severer cases, as has already been emphasized, it is anorexia rather than vomiting which is the most troublesome symptom. In some instances there is complete lack of appetite, even for breakfast or the beginning of a meal, and this develops into real refusal of food. If food is pressed on the child there is vomiting. Should the increase in the diet be made very gradually, the vomiting may be postponed for a day or two, but careful observation will show that, however the character of the food be modified, there is a definite limit to the amount of it which can be tolerated. The refusal of food may be quite as severe as in the untreated case of cœliac disease, and the atmosphere of the nursery at meal-time may be much the same. The gastromegaly child may show all the freaks of misbehaviour over

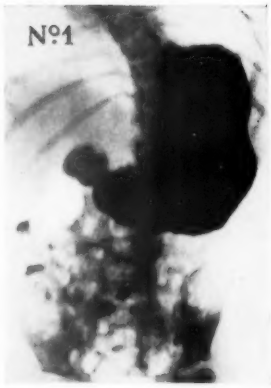
TABLE 2.

Early histories in present series.

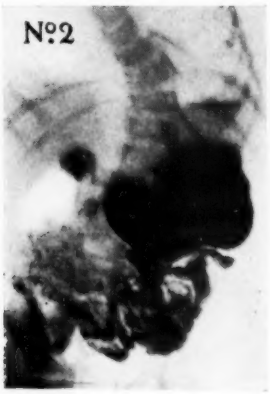
Case No.	Age of earliest symptoms.	Age when first seen (years).	Earliest symptoms.
1	From birth	$2\frac{1}{2}$	Anorexia, malnutrition, vomiting.
2	From birth	5	Anorexia, vomiting, malnutrition.
3	From birth	$5\frac{1}{2}$	Anorexia, vomiting, malnutrition.
4	Before 11 months	4	Malnutrition, diarrhœa.
5	Nine months	$2\frac{1}{2}$	Enlarged abdomen.
6	? from birth	6	Anorexia.
7	? from birth	8	Anorexia.
8	?	12	Misshapen abdomen, vomiting.
9	Sixteen months	$2\frac{1}{2}$	Enlarged abdomen.

food that are seen in cœliac disease and are so apt to lead to a diagnosis of nervous anorexia. In the milder cases the anorexia, although very persistent, is less spectacular. The absence of appetite is not due to the occurrence of pain after food, for of this there is very little : doubtless it is to be explained as the result of the gastric stasis which is present in all cases, and the gastritis which may be associated with it in the worst cases.

Vomiting. In the early stages the vomiting occurs sporadically after food, is projectile in type and copious in amount. Later it tends to occur in attacks and so, like any other recurrent vomiting in children, can be described as ' cyclical.' This alteration is due in part, no doubt, to the increase in the size of the stomach and improved capacity for storing food in it, but to a large extent it seems the result of the régime adopted. Where the child is fed down to its diminished appetite, vomiting can be avoided : where attempts are made to press the food, vomiting will occur.



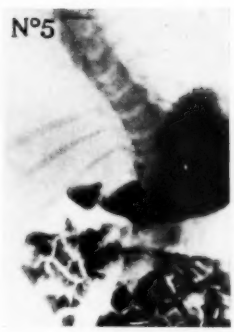
10 minutes, prone.



20 minutes, erect.

20 minutes, erect.

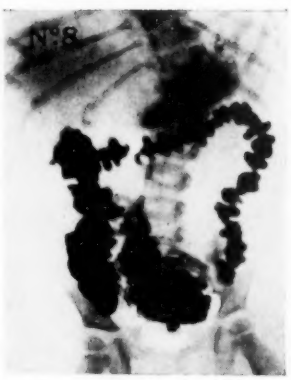
2 hours, erect.



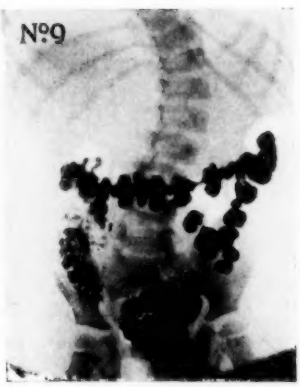
2 hours, prone.

3½ hours, prone.

3½ hours, lateral.



7 hours, prone.



24 hours, erect.

FIG. 1. CASE 1 : OPAQUE MEAL.

The vomit is large in amount, forcible in character, and contains food and usually mucus. Bile may be recognizable in it, but this is by no means invariable ; its colour, as Frank[22] has noted, may be lost by dilution. Careful observation of the vomiting in relation to the nature and time of previous meals, will establish the presence of gastric delay.

An attack of vomiting may come on suddenly or be preceded by a day or two of premonitory symptoms, such as increase in the coating of the tongue and in the refusal of food. Persistent hiccup may also give warning of an attack. Immediately before the sickness starts there may be abdominal pain. With the onset of the vomiting, there is fever, drowsiness, dehydration and rapid loss of weight. Almost at once diarrhœa occurs, and the stools contain mucus which, from its sodden appearance, may be taken to come from the stomach. The vomiting, owing to the unavoidable starvation, soon subsides, and the child is quickly rid of its toxic symptoms. The lost weight, however, is only regained slowly.

During the attack of vomiting, therefore, there is evidence of considerable toxæmia, from which at other times these cases are for the most part free. Duodenal stasis is known to produce severe toxicosis ; but in the attacks described here, although there is no question of the poisoning of the child, there seems nothing sufficiently peculiar to distinguish them as of duodenal origin.

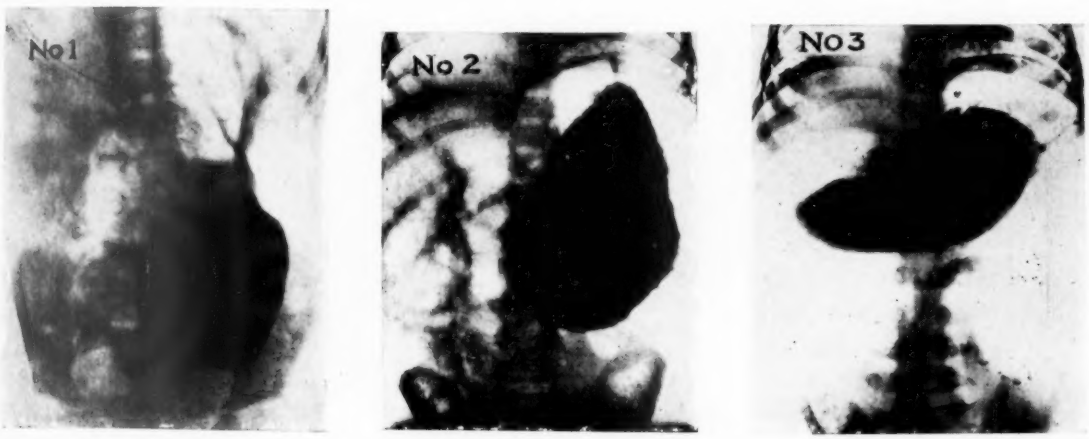
TABLE 3.

FAT ANALYSES OF FÆCES IN PRESENT SERIES. (DR. H. PERKINS.)

Case No.	Date.	Fat in dried fæces.				Diet.	Remarks.
		Total per cent.	Per cent. of fæcal fat.				
			Soaps.	Free fatty acids.	Neutral fat.		
1	10 : 7 : 28	10·4	69·2	27·0	3·8	Low fat	Undigested starch.
	6 : 12 : 28	24·0	92·6	5·0	2·4	Half fat	
	31 : 12 : 28	16·8	62·0	21·4	16·6	Half fat	
	6 : 3 : 29	No apparent excess				Full fat	Undigested starch.
	6 : 6 : 29	No apparent excess				Full fat	After operation.
	14 : 9 : 29	No apparent excess				Full fat	After operation.
2	22 : 10 : 29	59·2	48·0	41·2	10·8	Full fat	
	3 : 11 : 29	13·2	51·5	30·3	18·2	Low fat	
	19 : 2 : 30	16·0	57·5	40·0	2·5	Low fat	After operation.
4	1 : 12 : 29	23·8	58·2	34·3	7·5	Full fat	Indicanuria.
6	22 : 11 : 29	22·0	60·0	29·1	10·9	Full fat	Undigested starch.
7	7 : 1 : 26	20·0	52·9	37·3	9·8	Full fat	
9	4 : 9 : 25	46·2	34·9	30·6	34·5	Full fat	Indicanuria.
	22 : 2 : 26	37·2	29·2	55·6	15·2	Half fat	
	23 : 6 : 26	23·2	65·5	24·2	10·3	Low fat	
	5 : 12 : 27	19·2	58·3	37·5	4·2	Low fat	

In the only case in which careful watch was kept for the occurrence of acetonuria (Case 1), none was found before the vomiting declared itself.

The question of the mechanism by which vomiting is produced has been discussed in the section on pathogenesis.



5 minutes, erect. 10 minutes, erect. 2 hours, prone.

2 hours, lateral. 3½ hours, prone. 3½ hours, oblique.

FIG. 2 CASE 2 : OPAQUE MEAL.

GASTRITIS. Chronic gastritis is a rare disease in young children, but it is present in most of these cases of chronic duodenal obstruction. This is interesting in view of the fact that hypertrophic pyloric stenosis is the chief, if not the only, cause of really chronic gastritis in young babies. Doubtless in both conditions it is secondary to the gastric stasis. The signs of it are the persistently coated tongue, foul breath, mucus in the stools and vomitus.

Bowels. Where there is derangement of the bowels it is chiefly in the direction of constipation, due to the underfeeding from which these children as a whole suffer. During the vomiting attacks diarrhœa may supervene. This has already been sufficiently described in the preceding paragraph.

Two other abnormalities require mention. First, where the case is severe enough to be complicated by chronic gastritis, the motions may at any time contain mucus. The thickened, sodden appearance of this shows that it comes chiefly from the stomach. In Case 1, following the relief of the obstruction by gastro-jejunostomy, the signs of gastritis in time cleared up, after which the motions no longer contained mucus.

Secondly, fatty stools may at times be passed. This was the case in Cases 1 and 2, and again in Case 9 cœliac disease was suspected before the presence of obstruction was recognized. On low-fat diets the percentages of fæcal fat sank to figures lower than those usually obtained in cases of cœliac disease. Other cases showed normal percentages of fat in the stools. Failure in fat-splitting was found in only one of the specimens analysed (Table 3).

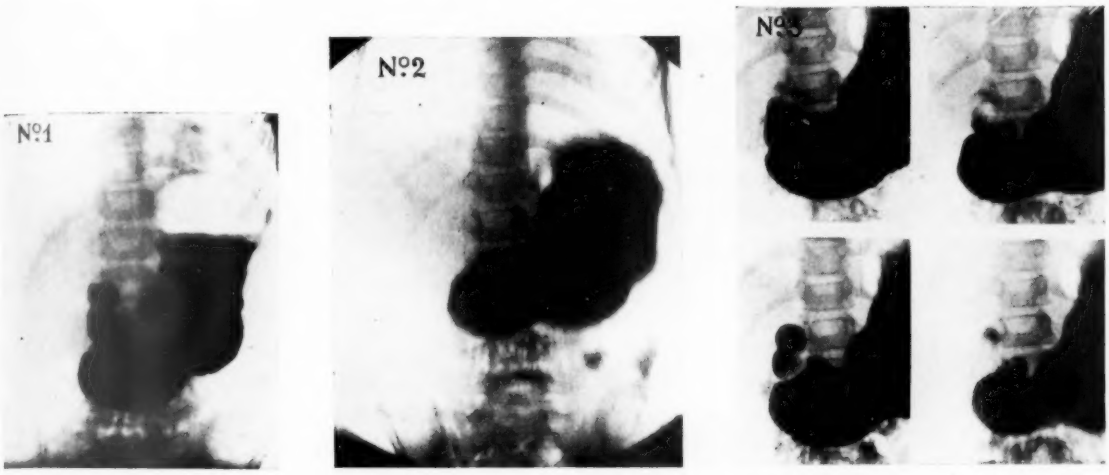
These observations suggest that there may be from time to time failure in fat-absorption owing to interference with the flow of bile out of the duodenum; and this impression receives some confirmation from the fact that in Cases 1 and 2, after gastro-jejunostomy, fatty stools were still occasionally passed. Unfortunately none of these particular specimens were submitted to chemical analysis.

It must be borne in mind that an excess of unabsorbed fat in the intestine may set up very considerable distension of the colon. The writer has reported[45] a case of cœliac disease in which megacolon was completely cured by a régime of fat-deprivation, in spite of the fact that for a long time the colon had been immensely dilated and had exhibited visible peristalsis. It is therefore possible that in cases of duodenal obstruction the voluminous appearance of the colon may be due to nothing more than defective fat-absorption.

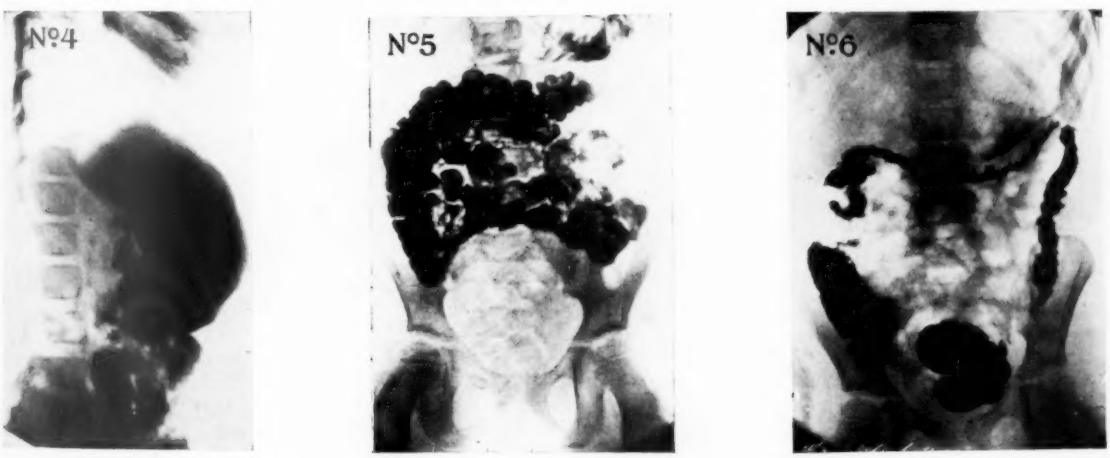
Pain. Pain in all the cases reported here was practically absent except immediately before actual vomiting. In this particular these cases differ from the reported examples of chronic duodenal ileus in adults and obstruction by band in children. For the most part they seem free even of discomfort, though doubtless both discomfort and pain could be brought about were food pressed on the child. Case 1, for instance, a most delicate boy, existing on a minimum of food, was a happy active little fellow, quite content to do anything except take food. In Case 2 there were complaints of discomfort in the abdomen, but here it appeared that the compensatory hypertrophy of the gastric wall had failed, and atony and some degree of gastroptosis had supervened. Where the tone of the stomach keeps good, and the child's limited capacity for food is respected, very little pain or discomfort seems to occur.

It is not altogether easy to understand why there is so little pain in these cases, although it must be admitted that in infants such a disorder as hypertrophic pyloric stenosis rarely gives rise to pain. Presumably it is because the persistent compression of the duodenum is so slight in degree. When the

distension of the stomach comes into play, the occlusion of the duodenum is increased, and pain and vomiting occur. Another possible explanation of the absence of pain is that the patency of the pylorus prevents a rise in the duodenal pressure.



5 minutes, erect. 10 minutes, prone. 20 minutes, prone; serial of cap.



25 minutes, lateral. 3 hours, prone. 7 hours, prone.

FIG. 3. CASE 3: OPAQUE MEAL.

HICCUP AND BORBORYGMUS. Hiccup is a very common symptom in all these cases although statements on its occurrence will seldom be volunteered. In most cases it occurs after meals, and in some it will recur after almost every meal. In those whose food capacity is very small, and who are therefore always living on the verge of vomiting, persistent hiccup may be a prelude to an attack of sickness. In practice hiccup forms a useful guide to the amount

of strain that is being put on the stomach by the particular diet in use. Doubtless the symptom depends on the distension of the stomach. In several cases excessive borborygmi were noticed to occur an hour or so after food.

NUTRITION AND GROWTH. The factor governing the under-development of these children is one of almost pure food-deprivation (underfeeding) and is accompanied by very little in the way of chronic toxæmia. In this they differ from the usual case of chronic indigestion in children, which is of intestinal origin and shows considerable chronic constitutional poisoning. In the present series only one child (Case 7) had the appearance of toxæmia, and he was suspected of having an unhealthy appendix. Most of the children, though thin, had clear skins, and several had quite pink complexions and showed nothing of the sallow unhealthy look of the 'intestinal child.' During the attacks of vomiting there is, as has been already mentioned, considerable toxæmia, but this soon passes off as the vomiting ceases. The only other sign of toxæmia which may be present is that of indicanuria, which may occur at any time when the bowels are loose.

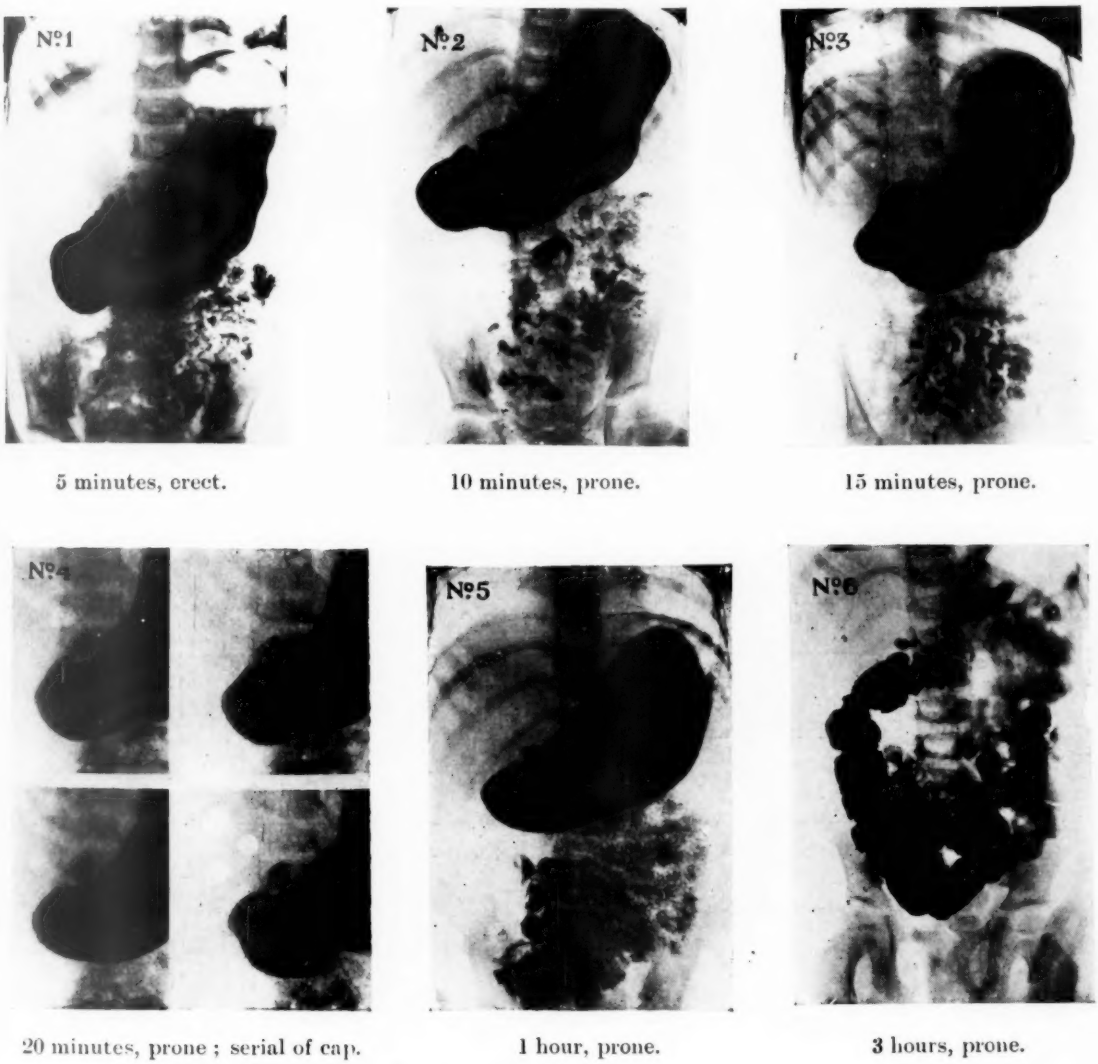
For these reasons, although in severe cases (such as Cases 1, 2, 3, and 4) the children have the appearance of smallness and frailty, their growth in height is not interfered with to the same extent as their growth in weight. They are not stunted to the same degree as is, for instance, the untreated case of cœliac disease. There the stunting of the growth is due to the combined effects of fat-deprivation and chronic toxæmia: in these duodenal cases it is due only to food-deprivation, and is more analogous to the condition found in œsophageal obstruction by Sheldon and Ogilvie[46].

It is worth mentioning that in the severer cases where there has been opportunity for close observation (Cases 1 and 2), alterations in weight of half a pound or more occur very easily in these children. It has been disappointing to note how little real improvement accompanies a gain of weight of a pound or two, though doubtless it is comforting to bear this in mind when the increase disappears in a day of upset and vomiting. It seems as though the best criterion of improvement in such difficult cases is their mental development, a gradual expansion in their interest in outside matters and an increased capacity for doing things for themselves.

PSYCHOLOGICAL CONDITION. In the severer cases where symptoms are present from the earliest months it is hardly possible for the psychological development of the child to proceed quite normally. A child who has never known what it is to want to take food, who perhaps has had all his life to have special food and to take all meals alone, can hardly fail to be influenced by such factors, so that the study of the psychology of these little underfed children is full of interest, particularly if the attempt is made to compare it with that of the child with cœliac disease. In some ways, especially over their tricks to get out of eating, they are rather alike, and yet there are differences. In the duodenal case there is, roughly speaking, pure under-development, while in the cœliac child there is an added element of psychological perversion; and it is tempting to suppose that the difference is due to the fact that in the one there is pure food-deprivation while in the other there is an added chronic toxæmia.

Physical signs.

The physical signs of this type of chronic duodenal compression are those of obstruction, and they cannot be distinguished from those of pyloric stenosis clinically except possibly by the recognition of bile in the vomit and the absence



5 minutes, erect. 10 minutes, prone. 15 minutes, prone.

20 minutes, prone ; serial of cap. 1 hour, prone. 3 hours, prone.

FIG. 4. CASE 4 : OPAQUE MEAL.

of a pyloric tumour. In none of the cases included in the present series was the duodenum obviously enlarged as in the group of infantile cases of megaduodenum mentioned earlier in this paper.

On inspection the enlargement of the abdomen is peculiar in that the protuberance affects the upper quadrants only. In more than one of the series it was the enlargement of the abdomen which was the cause of the child's coming under observation.

TABLE 4.

CLINICAL EVIDENCE OF OBSTRUCTION IN PRESENT SERIES.

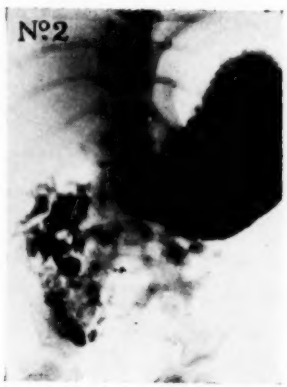
Case No.	Sex.	Clinical evidence of obstruction.		Remarks.
		Symptoms.	Physical signs.	
1	M	Anorexia, vomiting, hiccup, gastritis.	Enlarged stomach, vomiting projectile, visible peristalsis, palpable stomach, splashing.	Operation.
2	M	Anorexia, vomiting, hiccup, gastritis.	Enlarged stomach, vomiting projectile, visible peristalsis, palpable stomach, splashing.	Operation.
3	F	Anorexia, vomiting, hiccup, gastritis.	Enlarged stomach, vomiting projectile, visible peristalsis, palpable stomach, splashing.	Improving when first seen.
4	M	Anorexia, gastritis, (diarrhœa).	Enlarged stomach, visible peristalsis, palpable stomach, splashing.	Improved with medical treatment.
5	M	(Enlarged abdomen).	Enlarged stomach, visible peristalsis, palpable stomach, splashing.	No symptoms, progress good.
6	M	Anorexia, occ. vomg., hiccup, flatulence (asthma).	Enlarged stomach, vomiting projectile, visible peristalsis, palpable stomach, splashing.	Progressed slowly with medical treatment.
7	M	Anorexia, occ. vomg., hiccup.	Enlarged stomach, visible peristalsis, splashing.	Improving when first seen: progress good.
8	M	Vomiting (enlarged abdomen).	Enlarged stomach, splashing.	Improving when first seen: progress good.
9	F	Flatulence (enlarged abdomen).	Enlarged stomach, visible peristalsis (megacolon).	Progressed well with medical treatment.

It is essential that the abdomen should be examined when the child is lying on its back. Then, frequently, the outline of the stomach can be seen quite distinctly and its size, shape and position show that the organ is in a state of tonicity quite unlike the dropped flabby stomach of atony. The greater curvature crosses the middle line an inch or more above the level of the umbilicus; and often the body of the stomach extends well over to the right, covering the pyloric region as in hypertrophic pyloric stenosis. X-ray examination confirms this in many cases.

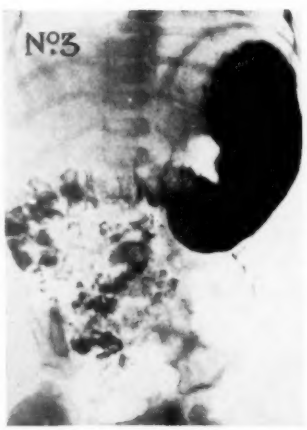
The size of the stomach can be determined not only by inspection, but also by the combined method of auscultation and light direct percussion. Splashing, which can be elicited at almost any time, proves that the dilated viscus is stomach and not colon. Hypertrophy of the wall of the stomach can be detected by palpation with assurance in a marked case in a thin child: in others it may be suspected.

Visible gastric peristalsis is, of course, the accepted sign of obstruction to the outflow of the gastric contents and hypertrophy of the musculature of the gastric wall. In these cases the peristaltic wave comes out from under cover of the left ribs and passes across to the right just as in the pyloric stenosis of infants. The three waves, all visible at once and so significant of hypertrophy of the stomach wall, are often well seen. Visible gastric peristalsis

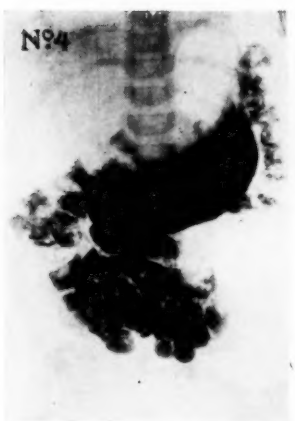
5 minutes, prone.



30 minutes, prone.



1 hour, prone.



3½ hours, prone.

FIG. 5 CASE 5 : OPAQUE MEAL.

has been seen in all the cases of this series except one (Case 8). In four (Cases 1, 5, 6 and 7) it was observed in routine examination of the child on the first occasion that it was overhauled. In Cases 2 and 4 it was observed after the children had been fed, but without inflation of the stomach. In Cases 3 and 9 it was observed only after the stomach had been inflated with a draught of soda water, but in both instances the peristalsis then was very well marked. It must be mentioned that both these children were seen only occasionally

and in consulting-room practice, so that it was not possible to test them after meals. Doubtless peristalsis would have been visible under such circumstances. In order to test the reliability of the inflation test in children it has been carried out in several thin children as controls, but the appearance of the stomachs in these cases has been quite different and no visible peristalsis has been seen.

Radiographic examination. In the present series of cases the value of the radiographic examinations has been confined to two points in diagnosis. First, it confirms the presence of obstruction high in the alimentary tract by demonstrating the enlargement of the stomach, suggesting hypertrophy of the gastric musculature, and proving the presence of gastric stasis. Secondly, it usually demonstrates satisfactorily that the site of the obstruction is not in the stomach or at the pylorus. Such assistance in diagnosis is of great value, but further than this X-ray examinations have not helped in this series: in no instance has there been obtained such direct evidence of duodenal obstruction as stasis, enlargement or cogwheel indentation affecting the duodenum.

That the distal parts of the duodenum do not show in the X-ray examinations of any of the present cases cannot be taken to negative the diagnosis of duodenal compression of the type dealt with here. The two cases (Cases 1 and 2) which showed dilatation and hypertrophy of the duodenum at operation, failed like all the others to display the distal parts of the duodenum under the X-rays. In another suspected case (not included in this series) an attempt was made to obtain a shadow of the whole of the duodenum by introducing barium cream through a duodenal tube. This manœuvre led to the satisfactory display of the duodenal cap, but the rest of the duodenum remained unseen.

The type of duodenal compression in these cases, as has been explained in the section on pathogenesis, is pictured as a mild chronic (persistent) compression, with intermittent periods of increased obstruction due to the action of the accessory obstructive factors, particularly distension of the stomach or colon. It may be that if the radiographic examination were to be made when the obstruction is accentuated, as during a bout of vomiting, the duodenum would show as distended throughout its course. Foucar found this to be so in one of his cases where the duodenum was compressed by a volvulus (see Appendix B). During a quiescent stage the duodenum appeared normal under the X-rays, but during an attack of vomiting it could be seen as enlarged throughout its length. No opportunity of examining any of the present cases radiographically during a bout of vomiting has occurred.

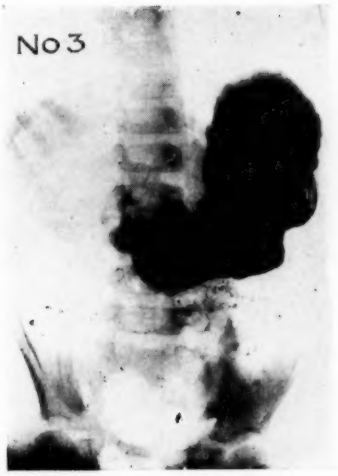
It must also be remembered that the condition of the duodenum as found at operation in Cases 1 and 2 was one of comparative enlargement only. It was large for a small child, but in neither case was it larger than that of a normal adult. This probably explains why the distal parts of it do not show in the radiological examination of these cases.

To get even such results as have been obtained here there are certain fallacies to be avoided and precautions to be taken.

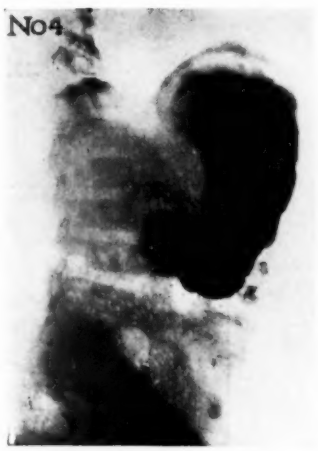
(1) If the meal given is too small or too fluid, the size of the stomach may be masked and the occurrence of gastric stasis missed. The same results may

5 minutes, erect.

5 minutes, lateral.

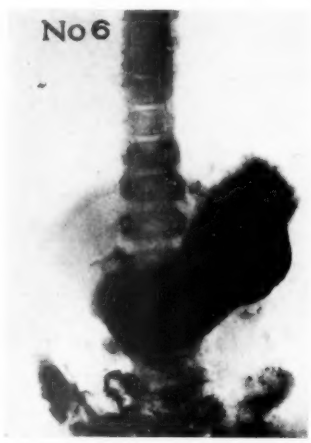


10 minutes, prone.



15 minutes, oblique.

1¼ hours, oblique.



1½ hours, prone.

4¾ hours, oblique.

4¾ hours, prone.

FIG. 6. CASE 6 : OPAQUE MEAL.

come from the use of a meal which does not contain food for digestion, as for example, a watery suspension of barium in tragacanth. Contrariwise, too large a meal may give rise to a false impression of gastromegaly, though such a fallacy is usually apparent to the experienced radiologist.

(2) The use of a standard barium meal is desirable, and such a meal was used in the examination of Cases 1 (second and third examinations), 2, 3 (second examination), 4, 5, 6 and 8. A somewhat arbitrary variation in the amount of the meal administered was made according to the age of the patient. The meal should be given after a sufficiently long period of fasting to ensure an empty stomach.

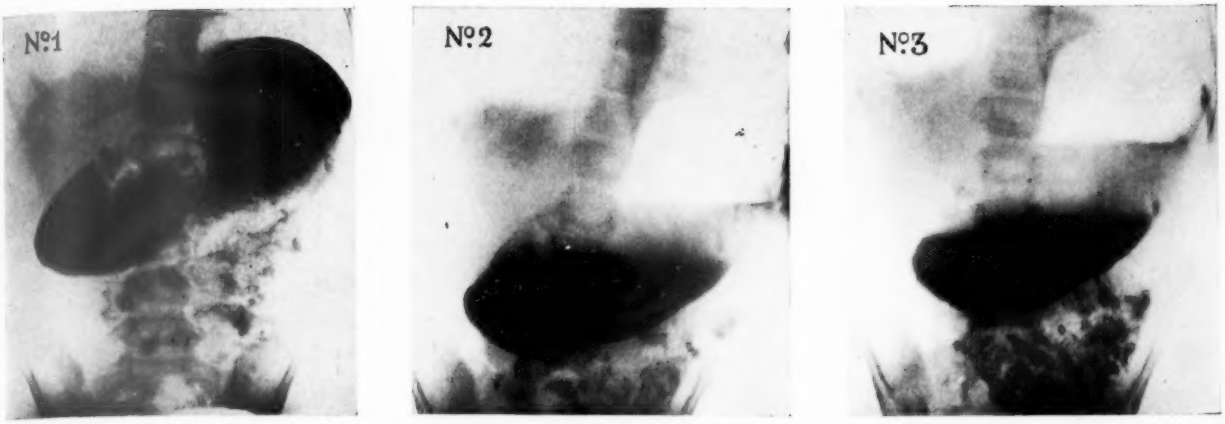
TABLE 5.

RADIOGRAPHIC EVIDENCE OF OBSTRUCTION IN PRESENT SERIES.

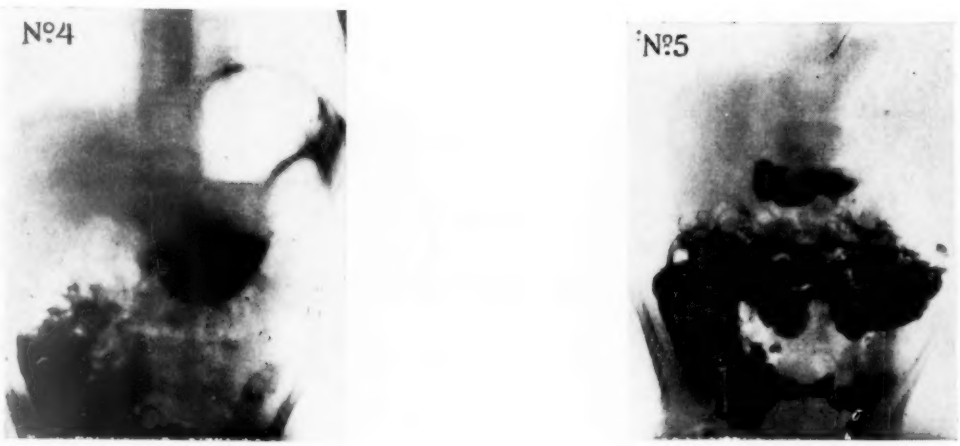
Case No.	Enlargement of stomach.	Hyper-peristalsis.	Emptying time.	Duodenum.		Remarks.
				Cap.	2nd and 3rd parts.	
1	Enlarged	Present	Increased	Normal	Not seen	
2	Much enlarged	Not seen	Much increased	Normal	Not seen	
3	Doubtful	Suspected	Not increased	Normal	Not seen	
4	Enlarged	Present	Increased	Normal	Not seen	
5	Much enlarged	Present	Increased	Normal	Not seen	
6	Enlarged	Present	Increased	Irregular	Not seen	? Cap imperfectly filled.
7	Much enlarged	—	Increased	Irregular	Not seen	? Cap imperfectly filled.
8	Enlarged	—	Increased	Normal	Not seen	Colon voluminous
9	Much enlarged	Present	Increased	Not seen	Not seen	Megacolon.

(3) In many instances the enlargement of the stomach is so great as to hide the duodenal cap in an antero-posterior view. This difficulty may be overcome by employing an oblique or semi-oblique exposure. In other cases a preliminary course of dieting may reduce the size of the stomach sufficiently to expose the duodenal cap in the antero-posterior view.

(4) The size of the duodenal cap is so variable within physiological limits that it is not safe to diagnose obstruction at the lower end of the duodenum unless the enlargement of the cap is greater than that seen in any of the present cases. In many instances the size of the cap appeared to be in normal proportion to the size of the stomach, and since the stomach was enlarged it might be argued that the cap was also enlarged. But in other instances the duodenal cap at the time of the examination appeared not at all enlarged, and so no definite evidence of duodenal obstruction has been accepted in these cases from a consideration of the size of the duodenal cap.



5 minutes, prone. 10 minutes, erect. 20 minutes, erect.



2½ hours, erect. 3½ hours, prone.

24 hours, prone. 48 hours, prone.

FIG. 7 CASE 7: OPAQUE MEAL.

Diagnosis.

The diagnosis of chronic duodenal obstruction in these cases is reached in two stages: first, by the clinical and radiographic evidence of obstruction at a high level in the alimentary tract; and secondly, by the absence of radiographic evidence of obstruction in the stomach or at the pylorus. These points have received sufficient attention in the foregoing pages.

The causes of chronic duodenal obstruction in children may be divided into intrinsic and extrinsic groups.

Intrinsic. 1. CONGENITAL DUODENAL ATRESIA OR STENOSIS. This group has been the subject of papers by Cowell[47] (1911), Cautley[48] (1919), Cameron[49] (1925), and Sheldon[50] (1926), in the English literature. The type of obstruction may be (a) by membrane, perforated or imperforate; (b) by fibrous cord, pervious or impervious; and (c) by blind ending of the duodenum. Life in such cases is only rarely prolonged for more than two or three weeks. Exceptions to this rule have been reported by authors whose cases have lived (without operative relief) for some months: Smellie[51], 3 months; Keith[52], 9 months; v. Koos[53], 13 months; Buchanan[54], 18 months; Cautley[48], 18 months. It must also be mentioned that a few cases of duodenal stenosis, taken to be of congenital origin, have been found in adults (Silcock[55], Moore[56], Perry and Shaw[57], Curling[58], Terry and Kilgore[59]). It is, however, evident that, although congenital stenosis may give rise to megaduodenum, as in the cases reported by Jewesbury[32], and Variot and Cailliau[35], and thus simulate the severe type of duodenal ileus in infants (see Group 1, p. 86), there is no great resemblance between these cases of stenosis and the type of chronic duodenal ileus described in the present series of cases.

2. CICATRIZATION OF PEPTIC ULCER OF THE NEWBORN. The writer has met one instance of this cause of gastromegaly (see p. 133). It is, however, of great rarity and presents no difficulty in diagnosis with modern methods.

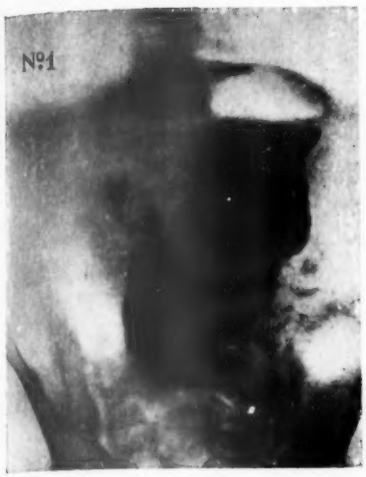
Extrinsic. In any case of chronic duodenal obstruction in a child more than a few weeks old, the cause of the obstruction is more likely to be extrinsic than intrinsic.

1. OBSTRUCTION BY PERITONEAL BAND. See below.

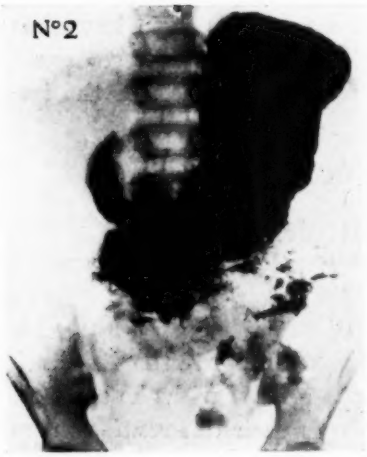
2. ARTERIO-MESENTERIC OBSTRUCTION. See below.

3. OBSTRUCTION BY VOLVULUS. Mention has already been made of the cases reported by Ombrédanne[40] and Foucar[42]. In the first, volvulus complicated duodenal ileus; and in the second a volvulus compressed the duodenum. Many other cases in which the volvulus, although due to developmental causes, is not directly concerned with the duodenum, have been recorded. Important papers on the subject are by Rixford[60] (1920), Dott[61] (1923), and Poynton[62] (1924). In these cases the attacks of obstructive vomiting are accompanied by great pain, and so differ from the condition described here.

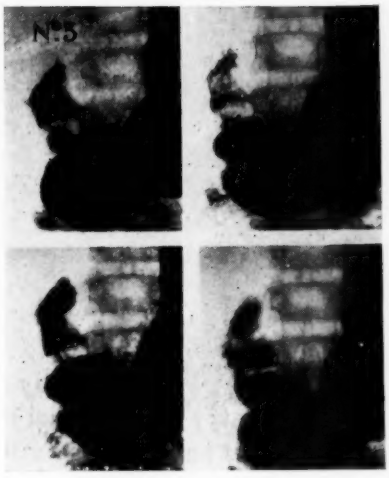
4. OBSTRUCTION BY DISEASED MESENTERY. This type of case as described by Foucar[42] (see Appendix B) simulates in the comparative latency of its obstructive symptoms the type of duodenal ileus described in this paper. In the present series, however, there has been no real difficulty in excluding active disease of the abdominal organs.



5 minutes, erect.

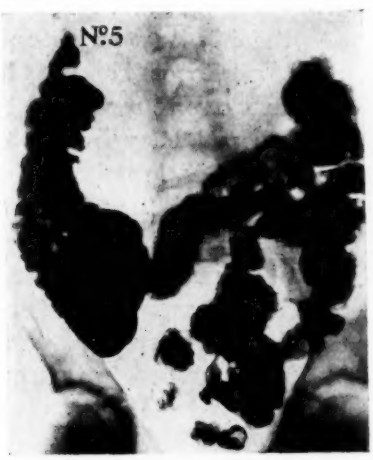


10 minutes, prone.

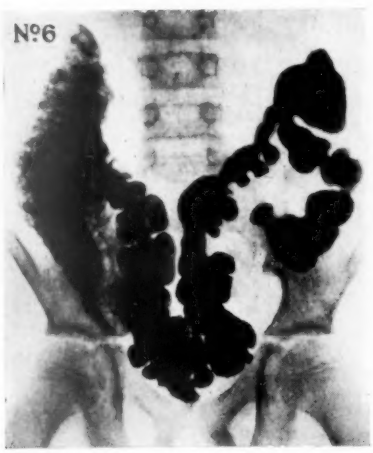


15 minutes, prone ; serial of cap.

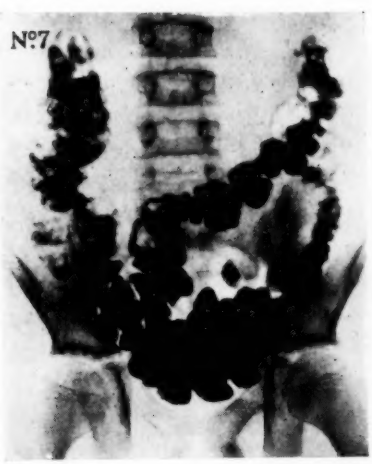
2 hours, prone.



7 hours, prone.



24 hours, erect.



48 hours, prone.

FIG. 8. CASE 8 : OPAQUE MEAL.

5. Obstruction by malformation of the mesentery or kinking of the duodenum. Cases of this type have been recorded by Waugh[43], and Judd and White[44] (see Appendix B). Denzer[63] has described congenital duodenal obstruction from mal-rotation of the intestine. Adult cases have been reported by Duval and Gatellier[64].

6. Obstruction by tumour. Hohlbaum's[36] case of compression of the duodenum by tumour in a new-born infant has been mentioned. Instances of this type have been recorded in adults by Bird[65].

7. Obstruction by pancreas. Compression of the duodenum by the pancreas has been described, the bowel being enclosed in a ring of pancreatic substance. The subject has been discussed by Anderson[66].

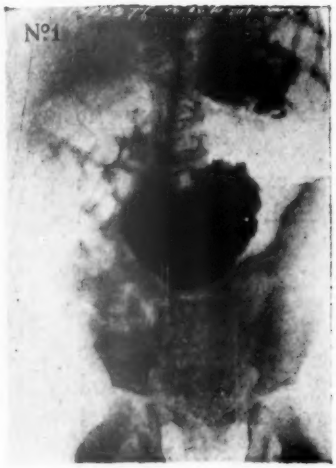
Undoubtedly the chief difficulty in the differential diagnosis of the present type of duodenal ileus is concerned with the cases of duodenal obstruction due to peritoneal bands. Nor is their differentiation unimportant. In the band cases operation is advisable and usually curative, whereas in the present type of duodenal ileus it would seem that operation is only a last resort and disappointing in its results unless a duodeno-jejunostomy is possible.

A fairly large number of cases in children of chronic duodenal obstruction due to peritoneal band has been described, and the literature on the subject is too extensive to reproduce here. Mention may be made of a recent paper by Higgins and Paterson[34] (1926). In most cases the bands are of congenital origin, and little is known of acquired periduodenitis in children. Clinically the cases of obstruction by band seem to fall into two groups. First, a very severe type giving rise to obstructive symptoms in early life, sometimes to megaduodenum, and so comparable to the first group of cases of duodenal ileus described above. Secondly, a type giving rise to dyspeptic symptoms, usually painful and interrupted by attacks of vomiting, in which the presence of an obstructive factor may be far less easy to recognize. This group is comparable to the second or adult type of chronic duodenal ileus. There appear, however, no cases reported of obstruction by band in which there is quite the same latent type of obstruction, causing very little pain and gradually improving spontaneously, as in the group of cases with which we are here concerned. But this is not to say that it is impossible for them to exist.

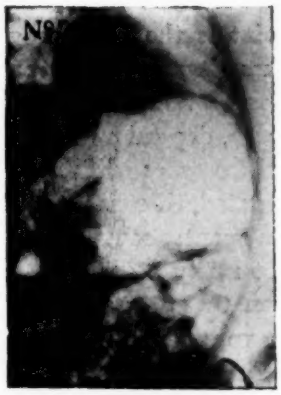
The differentiation of the cases described here and cases of duodenal obstruction by band cannot be said at the present time to be altogether clear; and although the writer has not taken such a line in his own cases, it could be held that in all such conditions the abdomen ought to be opened in order to exclude such an easily curable condition as obstruction by band.

Prognosis.

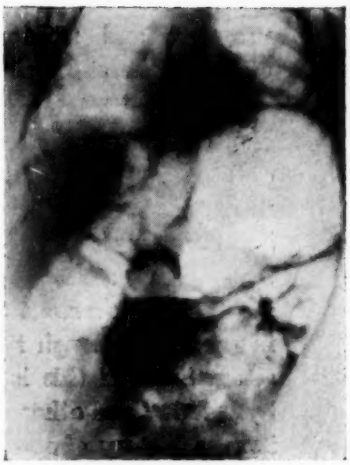
The immediate prognosis in these cases must depend largely on the degree of compression to which the duodenum is subjected, and this can only be estimated indirectly. One factor in this estimation will be the degree of gastric stasis as revealed by radiographic evidence and by careful observation of the relationship to past meals of material vomited. Another will be the child's



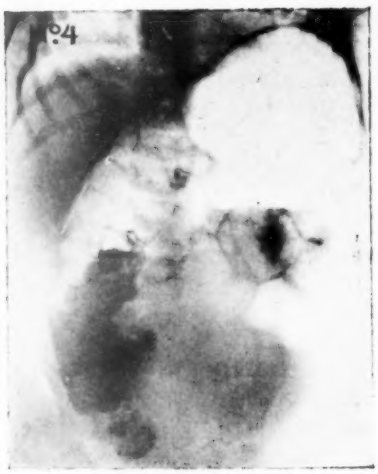
5 minutes, prone.



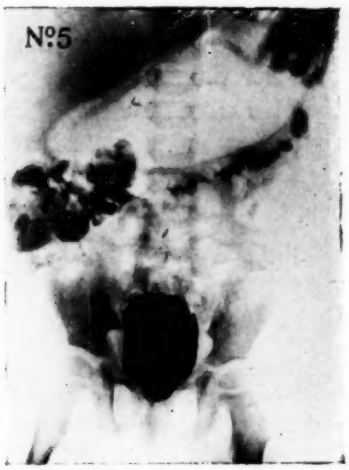
1½ hours, erect.



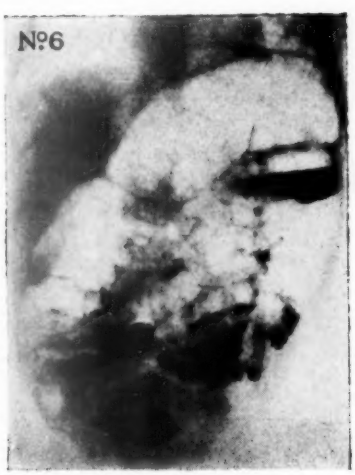
1¾ hours, erect.



8 hours, prone.



24 hours, prone.



36 hours, erect.

Barium enema, supine.

FIG. 9. CASE 9 : OPAQUE MEAL AND ENEMA.

capacity to develop gastric hypertrophy, and this will depend on its nutrition, progress, and freedom from intercurrent diseases. The development of atony of the stomach makes the outlook infinitely worse.

The most interesting point is the later prognosis, that is, whether the child, if it survives and makes slow progress, can be expected ultimately to outgrow its symptoms and lead a normal life. The views expressed on this point, as the writer is well aware, depend on the assumption that the diagnosis has been correctly made in those cases of this series which were not verified by operation : but if this is granted, there is no doubt that the tendency is for a gradual improvement to set in spontaneously some time after the age of about five years, although it may be many years before the vomiting attacks cease altogether. If this is so, then these cases correspond to the type observed by Wilkie[22] and others, who have noted in their adult cases of chronic duodenal ileus, a history of symptoms during childhood passing away in adolescence.

In connection with this point the following case is of interest. It is not included in the present series as the patient was not seen until the age of 29 years, and all direct evidence of obstruction to the duodenum is lacking. It must be a rare thing for a child to suffer from vomiting of the obstructive type for the first twelve years of life (and the patient's own statement was that his chief memory of his boyhood was ' being sick '), and then to recover spontaneously, and it is difficult to suggest any other organic condition to account for such a story except the type of chronic duodenal ileus described here.

P.K., male, aged 29 years. Born at full term, weighing 7¾ lb. Vomiting began at 10 weeks and continued with some diarrhœa and wasting until he was 4 months old. Then with gastric lavage it ceased, but at 5 months it began again. At this time refusal of food was very noticeable, and later became extreme. The mother described the difficulty in getting him to take food as ' incredible ' : he never got hungry however long he was starved, and if food were pressed on him he was sick. The vomiting was preceded by pain, and occurred chiefly in the evenings or at night. Hiccup was persistent, and the child habitually cross. The bowels tended to be loose. He took no solid food until he was 2 years old. His abdomen was large. He did not walk well until he was 5 years old, and at that time had the appearance of a child of 3 years.

At 5 years he began to improve, the vomiting growing less, though still considerable. From 7½ to 10 he was able to attend day-school. At 10 he went to a preparatory boarding-school, special provision being made for him to have his meals under supervision as they took from 1½ to 2 hours. He was then being sick about four times a term, but at 12 the vomiting ceased. He was kept at the same school until he was 17 owing to his frailty and small size. Puberty occurred at 19.

Seen at the age of 29 years, he was frail and light in appearance, and 5 ft. 1 in. in height. His father was 5½ feet, and his one brother the same. His mother was 5 ft. 1 in. He earned his living as an artist, and living mainly in the country contrived to keep well. He complained of transient attacks of dyspepsia of the gastric atonic type, and was seldom free of this trouble for long. There was no longer any vomiting.

X-ray examination (H.C.G.). The stomach was of good tone, peristalsis was well marked, and the emptying time normal (3 hours). The duodenal cap filled and emptied well. At 4 hours barium had reached the descending colon. At 20 hours the cæcum was filled. The cæcum was placed high, being at the level of the iliac crest in the prone and vertical positions. This might indicate incomplete rotation. The colon was probably rather larger than normal. The pituitary fossa was of normal size and shape.

The improvement in these cases which occurs so slowly can be hastened in some instances by medical treatment, but it is essentially a spontaneous improvement. The explanation of it is necessarily a matter of speculation. It is clearly not due merely to the development of an adequate compensatory hypertrophy of the gastric musculature. It is apparently the result of normal growth on the part of the child which eases the constriction at the duodeno-jejunal flexure.

As regards the possible dangers of the supervention of acute symptoms in these chronic cases, it has already been pointed out that the acute type of duodenal ileus does not seem to arise in children. Several of the cases of the present series had had operations performed under anæsthetics without the development of acute symptoms.

The outlook following operation for the relief of the obstruction in these cases is discussed in the section on treatment.

Treatment.

The factors underlying the treatment of these cases are as follows :— (1) there is chronic duodenal obstruction which will in the course of years lessen and disappear ; (2) it is sufficient to produce chronic gastric stasis and gastritis ; (3) it can be increased to the point of producing vomiting by distension of the stomach and possibly of the colon ; (4) there may be occasional interference with fat-absorption owing to the obstruction to the flow of bile from the duodenum.

MEDICAL TREATMENT. The chief indication is to give a diet which is small in volume, highly nutritious, not too rich in fat, and calculated to pass out of the stomach as quickly as possible. A pounded or sieved mixed diet suits these requirements best, and it should be given as dry as can be made palatable for the child. Meals should be separated from each other by as long intervals as possible. Drinks should only be given some hours after food. It is usually necessary to give lightly skimmed milk or to make use of a half-cream milk. Vichy water is useful as a drink or as a diluent to milk.

To prevent fermentation of the stagnant gastric contents, an alkaline antiseptic, such as glycothymoline in 15-minim doses, is of value. It may be given with a mixture of rhubarb and soda, using peppermint for a carminative. At times when the signs of gastritis have lessened, an acid mixture containing nux vomica may help the appetite. Castor oil has proved the most useful aperient in small children, and taxol tablets in older ones. If the intestine becomes distended, the colon should be washed out and measures taken to reduce the distension as soon as possible. The chief cause of intestinal distension seems to be fat indigestion. Gastric lavage and massage may be required.

Postural treatment has not the same value in these cases as in adults owing to the fact that they are not connected with visceroptosis in children. In Case 1, during the X-ray examination it was thought that the stomach emptied best in the prone position.

Surgical Treatment. As spontaneous improvement seems to set in after about the age of five years, surgical measures need only be considered in the severest cases in the younger children. Possibly in some older children lack of progress might persist owing to the development of a secondary atonicity of the stomach, but such cases would not be favourable for gastro-enterostomy.

In this type of case the enlargement of the duodenum is not sufficient to render a duodeno-jejunostomy a practical possibility, especially in a young, small and frail child. The alternative operation is that of gastro-jejunostomy. With an obstruction as low down as the duodeno-jejunal flexure this operation is at a disadvantage, and in adult cases of chronic duodenal ileus it has not been a success and has been abandoned in favour of duodeno-jejunostomy. It may be that in the congenital type of case described here it is too much to expect that any operation for the relief of obstruction will have the same happy and immediate effect as in an acquired obstruction. It must be remembered that these children have never, in the worst cases, looked upon food except with extreme distaste, and it is not a question of restoring a lost appetite but awaiting the development of a new faculty.

On these grounds it is clear that a gastro-enterostomy is to be regarded as the last resort, if all other measures have failed : and that lack of tone in the gastric musculature would be a still further contra-indication to the operation. From the experience of the two cases operated on in the present series it is possible to say that gastro-jejunostomy may be of use. In Case 1, where the tone of the gastric muscle was good, there is no doubt that the condition was alleviated, although the child's progress was slower than had been anticipated at the time of the operation. In Case 2 there was so much gastric atony that the possibility of benefit from gastro-jejunostomy was slight. On the whole the writer would not very willingly consent to operation on cases of this type unless something better than a gastro-enterostomy could be undertaken ; yet it seems clear that in very severe instances such an operation may benefit the child at least sufficiently to prevent the development of gastric atony behind the unrelieved obstruction.

Ablation of the pylorus, either at the time of the gastro-jejunostomy or later, has been stated by Downes (see Appendix B) to be of value. This is an operation which cannot be lightly undertaken in a small child, and it is not easy to see on what grounds it should be of benefit.

It is perhaps worth while mentioning here, since gastro-jejunostomy is so seldom performed in small children, that at any time after the operation a fatty feed such as full-cream milk, given on an empty stomach, may cause an immediate vomit of bile and mucus, evidently an evacuation of the gall-bladder and liver. Such an incident is followed later by the passage of a colourless stool.

Summary and conclusions.

Acute duodenal ileus does not appear to occur in children.

A summary of the previously published work on chronic duodenal ileus in children is given.

A series of cases showing enlargement and hypertrophy of the stomach (gastromegaly) is described. In them the symptoms date from birth or early life, and consist at first more of anorexia than of vomiting. Other symptoms are malnutrition, lack of growth, hiccup and borborygmus, mucus in the fæces from the presence of chronic gastritis, and occasional fatty stools. A peculiar feature is the comparative lack of pain in the abdomen except immediately preceding vomiting. Thus, the symptoms do not very readily raise the suspicion of organic obstruction to the evacuation of the stomach, but careful examination reveals the presence of obstruction, and skiagrams show that this does not arise in the stomach itself. Hence a diagnosis of gastromegaly from chronic duodenal obstruction is reached, and reasons are given for the view that the cases described owe their origin to chronic duodenal ileus.

There is in these cases a tendency for spontaneous improvement to occur during the later years of childhood. This recalls the fact which Wilkie and others have noted, that adult cases of chronic duodenal ileus may give a history of symptoms during childhood which pass off for many years during adolescence.

Other causes of gastromegaly from chronic duodenal obstruction are discussed.

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APPENDIX A.

Clinical reports of present series of cases.

Case 1. B.R., male, born February, 1926. 8th child in family of 8: all other children particularly strong. Born at 8½ months: birth weight 5¼ lb. From birth it was noted that he was frail and of feeble musculature.

During his first year he was not regarded as an ill child, but there was throughout great difficulty in getting him to take his bottles: it appeared as though he 'would not be bothered to take his feeds.' The amounts taken were very small and he displayed no appetite even for the beginning of his bottles. He remained small with flabby muscles. At the end of the first year he weighed 14¼ lb. There was no vomiting of significance at this time.

During the second year vomiting began to become troublesome. The first attack of any severity occurred at 13 months, but such attacks only became frequent after the age of 18 months. Careful observation during the ensuing months showed that vomiting occurred when attempts were made to increase the amount of food taken. The child took, even with difficulty, very small and inadequate meals, and when by pressure he was made to take more, the refusal of food would increase, the tongue would become more than usually coated, hiccup would be persistent, and after a few days, unless the diet were materially decreased in amount, a bout of vomiting would occur. The sickness usually started at night, preceded by discomfort or actual pain, the vomit being large in amount and projectile in type, and containing food and much mucus which was often bile-stained. During the attack of vomiting there was considerable toxæmia, with fever and rapid loss of weight from dehydration. The vomiting was quickly followed by diarrhœa with much mucus in the stools.

At 13 months scoliosis from muscular atony first appeared. This increased considerably in spite of the fact that the child was kept off his feet and did not walk until he was 26 months old. At 13 months he had 9 teeth, dentition being complete by the 26th month. At the end of his second year he weighed 18 lb. 10 oz.

During the third year the condition remained much the same. There was no increase in the amount of food which could be taken, so that the supply become increasingly inadequate for the child's growth.

The boy had been under the care of pædiatricians all his life and was first seen by the present writer in consultation in July, 1928, as a possible case of cœliac disease. He was then 2 years and 4 months old, and weighed 18½ lb. His height could not be taken owing to the scoliosis. It was noticeable that his complexion was beautifully pink, showing neither toxæmia nor anæmia. His limbs were very small and flabby; his buttocks atrophic. The abdomen was enlarged but chiefly in the upper part, and above the level of the umbilicus gastric peristaltic waves could be seen travelling from left to right across the abdomen. These had not been previously noted. Gastric splashing was easily elicited. Readings of the blood fat had been taken and were accounted high, (1·17, 1·15, and 1·51%).* The child was already on a low-fat diet, and analysis of the stool showed the percentage of fat to be 10·4%. The protein and fat appeared to be well digested, the starch of the food was less successfully digested.

First X-ray examination. In order to investigate the condition of the stomach a liquid opaque meal was given. This, although it demonstrated considerable enlargement of the stomach, failed to show any gastric stasis. The outlet of the stomach was not seen, being hidden behind the enlarged body of the organ. The diagnosis of cœliac disease was therefore set aside in favour of one of general enfeeblement of digestion, especially of vegetable starch. The presence of the visible gastric peristalsis remained unexplained.

The boy was not seen again until December, 1928, when he came under the care of the present writer. His condition was then much the same as it had been six months before. His weight was 19 lb. 7 oz. Gastric peristalsis could be seen after meals, and at other times there was much splashing in the stomach. The conclusion was therefore reached that there was obstruction to the emptying of the stomach which was of real moment, but that it came into

* Permission has kindly been granted to mention that this case (B.R.) figures in Table II of the paper on the blood fat in cœliac disease by Moncrieff and Payne.[67]

play chiefly, or perhaps only, when the stomach was distended. It was clearly necessary to obtain a radiographic picture of the exit of the stomach, and as in the first X-ray examination the pyloric region had been completely hidden by the enlarged body of the stomach, it was determined to reduce gastric distension as much as possible before a second radiographic series was taken. To this end the child was placed on a dry diet, with drinks between meals, daily aperient enemata to reduce possible stagnation, and gastric antiseptics to reduce fermentation. With these alterations there was an immediate improvement in the appetite, but this waned after two or three weeks although the feeding never again became quite so difficult as it had been at its worst. It is interesting that at this time, for the first time in his life, the boy would take a cup into his own hands for a drink.

Under this régime the gastric peristalsis was no more seen, splashing became rare, hiccup disappeared, and the child seemed happy and free from discomfort; but he was unable and unwilling to take enough food to allow for growth and development.

SECOND X-RAY EXAMINATION. (H.C.G.) On February 26th, 1929, the boy being now 3 years old, an opaque meal was given consisting of 2 oz. of Allenburys food, 2 oz. of barium, made up to 8 oz. with milk. Of this the child took 6 oz. (Fig. 1).

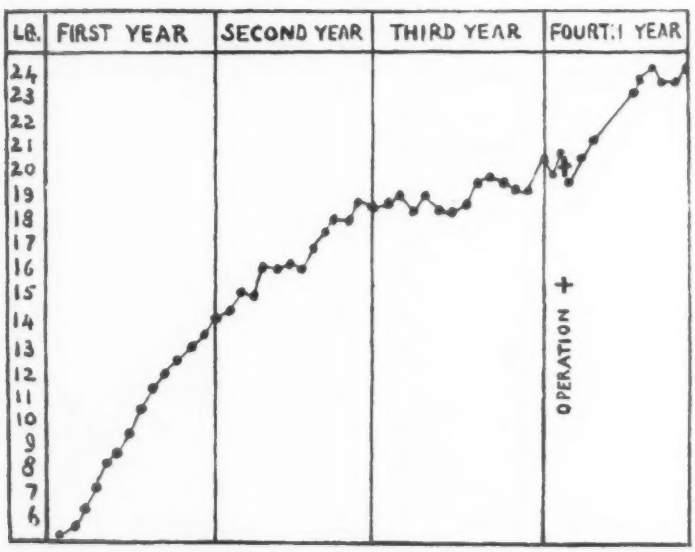

Chart showing weights at 4-weekly intervals in Case 1. The weight becomes stationary in the third year as the amount of food given cannot be increased to meet the increased need of the child.

The stomach was undoubtedly large, but of good tone and showing hyper-peristalsis. It took, however, much longer than the average at this age and with this meal to empty, there being a fasting residue of about 2 oz. at 3½ hours. There was also a residue at 4½ hours when food was permitted, which accounts for the presence of barium in the stomach in the films taken at 7 hours. The duodenum was judged to be normal, and a lateral view of the stomach and duodenum showed the anterior and posterior surfaces of both stomach and duodenal cap. The distal parts of the duodenum were not seen in any of the films. The passage through the colon presented no unusual features. At 24 hours the cæcum was practically empty, the bulk of the meal being in the transverse colon, sigmoid and rectum.

Manipulation and changes in posture showed that the stomach tended to deviate very much to the right when the child lay on his right side. Possibly the scoliosis facilitated this displacement. It was thought that in this position the emptying of the stomach would be delayed and that the prone position would be the best that could be adopted from this point of view.

Diagnosis. It was now clear that the delay in the emptying of the stomach was due to some obstruction high up in the alimentary tract. The clinical evidence of this was :— (1) gastric stasis, as shown by splashing, vomiting, hiccup, etc., (2) occasional visible gastric peristalsis, (3) palpation of the thickened gastric wall through the abdominal parietes, (4) presence of chronic gastritis, comparable to that found in the hypertrophic pyloric stenosis of infants, as shown by coated tongue, mucus in the vomit, persistent presence of large collections of sodden mucus in the stools. The second X-ray examination confirmed the presence of enlargement and hypertrophy of the stomach, but gave no indication of the nature or position of the obstruction, except that it was not in the stomach itself.

It was at this time very difficult to decide between two alternative diagnoses. If the obstructive factor were acting persistently, although increased by gastric distension, the diagnosis of duodenal obstruction, intrinsic or extrinsic, appeared most probable. If, on the other hand, the obstruction came into play only when the stomach was considerably distended, it might possibly be due to kinking in an enlarged stomach. Two points favoured the second alternative. First, that the first X-ray examination was taken to show no evidence of any obstruction at all. Secondly, that the only other case which the writer had recognized at that time (Case 7) had improved on measures directed towards preventing over-distension of the stomach.

It was therefore determined to persevere with the régime already described for a further two months, making five months in all. At the end of this time it was found that the amount of food which could be taken showed very little increase ; that 3 meals of about 6 oz., with a daily ration of more than 12 oz. but less than 18 oz. of fluid, were all that could be tolerated ; that if more than these amounts were given there would develop strenuous refusal of food, hiccup, and the other preludes to an attack of vomiting. Further, the passage of a tube 3½ hours after a meal still showed a large residue even at the end of 5 months of this strict régime. The boy's weight had slowly increased to 20 lb. 12 oz., a gain of 1 lb. 5 oz. in 5 months.

It therefore became evident that, although it was not certain that the obstruction was persistent, the amount of gastric distension necessary to produce obstructive symptoms was so small that the only hope of progress lay in operation for the relief of the obstruction. At the end of April, 1929, the boy was seen in consultation with Dr. R. Hutchison and Mr. Barrington Ward, both of whom agreed to the wisdom of operation.

Operation. Mr. Barrington Ward operated on May 6th, 1929. The abdomen was opened by a paramedian incision. The stomach was found to be considerably enlarged and its wall definitely hypertrophied. The pylorus was rather wide and dilated. The duodenum was dilated to about the breadth of two fingers : this dilatation affected the first and second parts of the duodenum, and the third part as well, as far as could be determined. The jejunum at the duodeno-jejunal junction was very small and collapsed, and was only about one-third the width of the third part of the duodenum. No definite bands were found at the junction, but the superior mesenteric artery and the mesentery formed a very tight band across the duodenum. The small intestine was small, empty and atrophic, and hung rather low. The transverse colon was normal. The ascending colon was fixed. The cæcum was, perhaps, a little high.

A posterior no-loop gastro-enterostomy was performed. After this was completed the jejunum seemed to be filling up to twice its original size. The abdomen was closed.

The condition found was therefore exactly comparable to the chronic duodenal ileus of adults.

Post-operative progress. The boy stood the operation well. The stomach was at once reduced in size, its lower border rising towards the epigastrium and feeling extraordinarily hard. The hypertrophy of the gastric wall gradually disappeared, and after many weeks the mucus disappeared from the stools, showing that the gastritis had passed off. Vomiting of food became very infrequent and, as it were, accidental. Thus there was clear evidence that the operation had relieved the obstruction to the evacuation of food from the stomach. On the other hand there were still signs that the flow of bile could be obstructed. At times the fat in the food was not well absorbed and the consequent intestinal distension would sometimes produce vomiting. Further, a fatty feed, such as a drink of full cream milk, given on an empty

stomach, would sometimes cause almost immediate vomiting of a large amount of bile and mucus, clearly from an evacuation of the gallbladder and liver. After such an event the next stool would be colourless.

Gradually considerable improvement occurred. The child's intelligence began to expand, his memory to improve, and it was noticeable that he responded to a change of air in a way that he had not done before. But the gain in weight was disappointingly slow, although an improvement on the almost stationary weight of the year before the operation (see Chart), and there was still great difficulty in getting enough food taken. It gradually became clear that the operation was not to be followed by the quick improvement that is the rule in such a condition as an acquired pyloric stenosis.

Four possible handicaps to progress existed. First, the unrelieved obstruction to the flow of bile. Secondly, the psychology of the child. It has to be remembered that the boy had never known what it was to be hungry, nor indeed anything other than extreme distaste for food. Further, owing to under-feeding and under-development, he behaved like an experienced baby, not easily led by reason. Thus he was extremely difficult to persuade to take food properly. Thirdly, poor tissue assimilation owing to the under-feeding which had existed from birth. Fourthly, possible atony of the stomach with gastric stasis persisting in spite of the operation.

3RD X-RAY EXAMINATION (H.C.G.). In February, 1930, nine months after the operation, a further X-ray examination was undertaken in order to investigate the last point. It was found that a 3-oz. feed of the standard meal was well held up by the gastric muscle and passed rapidly through the stoma. 48 hours later an 8-oz. feed consisting mainly of minced chicken was given. With this meal the tone of the muscle relaxed somewhat and the meal was held for about 2½ hours, but at the end of this time the stomach completely emptied through the stoma, although the duodenal cap was observed in one film.

This examination, therefore, showed that all the advantage that could be obtained by gastro-jejunostomy had accrued in this particular case, and that the comparatively slow progress depended on the other difficulties inherent in this condition. Doubtless complete recovery will ultimately take place, but it must be concluded that even in such a case as this, where everything seemed favourable and very skilled nursing was available for the child, gastro-jejunostomy does not offer any rapid cure.

Case 2. A.J.E., male, born October, 1924, and first seen (October 3, 1929), at the age of 5 years.

Full term child, weighing 8¾ lb. at birth. He was breast fed for 2 months. From birth he took his feeds slowly and never seemed ravenous for food, but he progressed well until the 7th week, when he weighed 12¼ lb. At this time he started to vomit, pumping up his food and crying as if in a good deal of pain. At 10 weeks he was admitted to the Hospital for Sick Children, Great Ormond Street, London, as a possible case of pyloric stenosis. The following notes on his condition during his stay there have been kindly supplied :—There was no distension of the abdomen, no visible peristalsis, and no palpable pyloric tumour. While in hospital there was some vomiting. Ultimately he gained weight well, though he still vomited occasionally, and after 6 weeks he was discharged with a diagnosis of 'indigestion.' After his discharge he attended for a time as an out-patient and had no more vomiting until he was 6 months old. Then it returned, but not seriously, and he gained well until the age of 10 months. At this age he had measles, followed by some debility, and the vomiting increased, but by giving him small frequent meals he made fair progress until he began to take solid food at the age of 18 months.

From this time his appetite was extraordinarily bad and for days together it was almost impossible to get him to take any food at all. Attacks of vomiting recurred every 3 or 4 weeks, when he vomited two or three times a day and refused all food. The attacks were accompanied by fever, drowsiness and rapid loss of weight : mucus was present in the stools. After an attack he would eat well for a day. Hiccup was a prominent symptom and might be a prelude to a vomiting attack.

At the age of 5 years he was said to be distinctly worse than he was at 3, eating even less than he did, and very languid and silent. He was thought to be getting thinner although still growing in height. The stools were usually constipated but occasionally large, pale and loose.

On October 3rd, 1929, he attended the Paddington Green Children's Hospital as an out-patient under Dr. Tallerman who admitted him for investigation under the care of the present writer on October 17th.

He was then 5 years old and a delicate, frail-looking boy, weighing 34 lb. 14 oz. He was extraordinarily silent, neither talking nor smiling, but sitting up in bed watching. He seemed to be very much the picture of a chronic invalid child. It was impossible to tempt him with any kind of special food, and he took the ordinary diet very poorly. His tongue was coated, and he sat usually holding his stomach as though in discomfort, though there seemed no real pain. The outline of the stomach could be seen under the abdominal wall reaching almost down to the level of the umbilicus, obviously larger than normal. Splashing was easily elicited. The wall of the stomach could be felt through the abdominal wall, suggesting hypertrophy, but no visible peristalsis was at first seen. On distending the stomach with a draught of soda water feeble but definite waves of gastric peristalsis were seen crossing the abdomen; later, peristalsis was seen after feeding without inflation of the stomach. The condition of the stomach suggested one of obstruction with secondary failure of the compensatory hypertrophy. On a diet containing full-cream milk the stools were pale and greasy and contained 59·2% of fat. On a low fat diet this figure sank to 13·2%. In both specimens the fæcal fat was adequately split.

X-RAY EXAMINATION (H.C.G.). On November 7th an X-ray examination showed a large dilated and atonic stomach, very deficient in peristalsis with much gastric delay, there being a residue of practically half the meal at 3½ hours. The duodenal cap appeared normal. No evidence of any organic cause for the gastric stasis was discovered (Fig. 2).

DIAGNOSIS. From its similarity to Case 1 it was not difficult to surmise that there was duodenal obstruction from arterio-mesenteric ileus, but that in this case the compensatory hypertrophy of the gastric musculature had failed. As there seemed no chance of getting improvement here and the child's condition has been steadily deteriorating according to the parents' account, it was determined to operate for the relief of the obstruction.

OPERATION. On November 22nd, 1929, the abdomen was opened by Mr. Shattock in the right paramedian line. A small amount of clear fluid escaped, the origin of which remained unexplained. No signs of tuberculosis were found in the peritoneum, mesentery or glands. The stomach was large, dilated and flabby, but its wall appeared thickened. The duodenum was dilated and hypertrophied, appearing like an adult duodenum. This abnormality was present as far as the duodenum could be traced (first and second parts). The jejunum was normal in size. Two small peritoneal bands or thickenings were found but were not obstructing the bowel: they ran upwards from each side of the pylorus. The colon was distended but was not abnormally movable. The cæcum was not high in position, and the hepatic flexure appeared normal. The condition was taken to be one of chronic duodenal ileus, and a posterior gastro-jejunostomy performed.

COURSE. In the three months following operation only slight progress was made, though periods of increased interest in surroundings, improved appetite and gain of weight were seen. It was evident that, although the stomach had diminished in size since the operation, there was sufficient lack of tone in it to be a serious handicap. The stools at times were fatty, and only half-cream milk could be tolerated. Any distension of the intestine quickly produced vomiting. The psychological state made it very difficult to appreciate the true value of his symptoms. At Christmas, for instance, he eat well and gained weight, but at a visit from his parents he would lapse into complete apathy and silence, and could not (apparently) summon up strength enough even to wave good-bye to them. Thus it was very difficult to judge how much of the refusal of food was due to his mental attitude, and how much to unrelieved gastric stasis from gastric atony.

Case 3. M.M., female, born June 1923. First seen at the age of 5½ years (December, 1928), referrred by Dr. Elder of Mansfield. Brief notes are available from birth, but weight records of infancy are missing.

Birth weight was 4 lb. From birth it was very difficult to get the child to take food properly. In addition there was fairly frequent sickness, the vomit being often definitely projectile. Diarrhœa, with stools containing mucus and sometimes blood, was associated with the vomiting. Of the three symptoms, refusal of food, vomiting and diarrhœa, the most troublesome was the

first. The taking of food seemed to cause discomfort and so food was obstinately refused. The baby was habitually cross. Hiccup was a prominent symptom. At 3 months her weight was 7 lb. 13 oz. After 6 months she began to get a little better, and still further improvement occurred between 18 and 24 months, attributed to the change on to solid food.

Contemporary notes during the 2nd, 3rd and 4th years constantly refer to the difficulties in getting the child to eat. Gradually the vomiting settled down into attacks typical of gastric stasis. The sickness would occur in the evening, preceded by increased refusal of food and extra coating of the tongue. The vomit was large, contained mucus and altered food, and the sickness was followed by loose motions containing mucus. During these years growth in weight and height were slow. At 5 years she weighed 38 lb.

First X-ray examination. (The nature and the quantity of the meal used are not known.) In November, 1928, an X-ray examination showed a very large stomach covering over the pyloric region. The tone of the stomach appeared normal and peristalsis did not appear to be active at the time when the films were taken. The emptying time of the stomach was, however, only 2 hours.

The girl was first seen on December 28th, 1928. She was then 5½ years old and weighed 36½ lb. Her height was 43 in. For the last six weeks, following the X-ray examination, she had been placed on a dry diet and had had no vomiting. She had, however, lost ¾ lb. On examination nothing abnormal was found except in the region of the stomach. This organ appeared enlarged, splashing was easily obtained, but no peristalsis was seen even after a draught of cold water had been taken. In September, 1929, however, well-marked peristalsis was seen following distension of the stomach by a draught of soda water. The feel of the stomach through the abdominal wall suggested hypertrophy of its walls. The régime of a dry diet was continued and made more stringent.

On March 19th, 1929, the child was seen again. She had continued free of vomiting and had now lost all nausea. Her weight had increased to 38 lb. 12 oz., and she had grown ½-in. in height since December.

Second X-ray examination (H.C.G.). At this time a second X-ray examination was made with an opaque meal consisting of 2 oz. of Allenburys food, 3 oz. of barium, made up to 12 oz. with milk. The stomach was regarded as average in size: its tone good and peristalsis suggestive of hypertrophy. The emptying time was normal: at 3 hours only a trace of the meal remained in the stomach. Nothing abnormal was detected in the stomach, and the duodenal cap filled and emptied normally. At 3 hours the meal had reached the middle of the transverse colon. At 7 hours the sigmoid was full, and at 24 hours the whole of the meal had passed. The bowels had acted twice in the interval, although no aperient had been used. The colon appeared average in length and size (Fig. 3). In connection with this result it must be remembered that the child had been completely free of all symptoms for 4 months before the X-ray examination was made.

In September, 1929, the girl was seen again. She was now 6¼ years old: weight 38 lb., and height 44 in. She had had an attack of scarlatina which had delayed progress, but there had been no vomiting at all. Her appetite showed distinct improvement. By administering a draught of soda water well marked gastric peristalsis became visible.

In March, 1930, her weight was 42½ lb., and her height 45 in. She had had whooping-cough and chicken-pox. She had remained free of vomiting; her appetite, though still subnormal, had improved. Visible gastric peristalsis was elicited as before.

Diagnosis. In view of the history of the case and its similarity to Cases 1 and 2 it was difficult to doubt that some obstruction was present to the emptying of the stomach. The X-ray examination did not give satisfactory confirmation of this view, probably because spontaneous improvement had already set in. Definite proof of obstruction was ultimately obtained by the production of visible gastric peristalsis after inflation of the stomach by soda water. It is therefore suggested that obstruction was due to chronic duodenal ileus.

Course. For 16 months on a dry diet there had been no vomiting and the appetite had shown improvement. It was clearly right to continue treatment on the same lines.

Case 4. H.D., male, aged 4 years. Brought for malnutrition and diarrhœa. First seen November 28th, 1928. Lived at Pau. History of the first 11 months of life was not obtainable,

but at this age he was frail and weighed 14 lb. At 2 years he was stronger and until 3 he got on fairly well, but was subject to attacks of diarrhœa, the motions being large, pale and offensive. Since the age of 3 years his progress has been very slight. His diet has had to be rigorously restricted or diarrhœa would supervene. He was regarded as an obscure case of indigestion, and on the restricted diet his appetite was good. There was hiccup but no vomiting.

At 4 years of age he weighed 28 lb. and was of normal height, but very frail in appearance. His tongue was thickly coated and his breath foul. His abdomen was large in its upper part and the outline of an enlarged stomach could be clearly seen. The wall of the stomach felt thickened and gastric peristalsis was visible. Splashing could be elicited at any time. Examination of the stool showed that the food was well digested: total fat = 26·8% of the dried fæces. The urine was normal except for a great excess of indican.

The boy was admitted to a nursing-home. His diet was increased and he eat readily. He was given pounded food with drinks between meals.

X-RAY EXAMINATION (H.C.G.). On December 1st he was given an opaque meal consisting of 2 oz. of Allenburys food with chocolate, made up to 8 oz. with milk. The stomach was found to be considerably enlarged, tending to hide the outlet. The tone was good, peristalsis was powerful and in one film suggested hyper-peristalsis. There was a small residue at 3½ hours. The duodenal cap, seen in the semi-oblique position, was normal, and no abnormality other than the enlargement of the stomach was found. The meal was not traced through the intestine (Fig. 4).

DIAGNOSIS. Mild obstruction to the emptying of the stomach, probably due to chronic duodenal ileus.

COURSE. During the fortnight that he was under observation the boy gained 3½ lb., and except for some hiccup he was free of symptoms. He had, however, been previously on an extremely low diet, which accounted for his rapid gain. In the next 14 weeks, at the seaside he gained 4 lb. 10 oz.

Case 5. D.M., male, aged 2 years and 4 months when first seen (May 29, 1929). Referred to Paddington Green Children's Hospital for enlarged abdomen by Dr. Broadbridge, as possible Hirschsprung's disease.

Full term, weighing 8 lb at birth. First tooth cut at 6 months; began to walk at 15 months. Has one sister aged 13 years. He was breast fed at first.

Towards the end of the first year, probably about the 9th month, the abdomen was noticed to be big. The enlargement tended to increase, but as the boy was quite well except for attacks of bronchitis, he was not taken to any doctor until quite recently. Throughout there has been no vomiting and the appetite has been good, though he required coaxing over his food at times. Weight has been maintained. Bowels regular.

The child was fast asleep when first seen in out-patient department. The upper part of the abdomen protruded and across it were passing large peristaltic waves from under the left ribs to the right. The stomach was enlarged, reaching the umbilical level, and its wall could be felt evidently hypertrophied. Splashing was easily elicited. No tumour was felt.

The boy was admitted and under observation he eat well and was not sick. His weight was 25 lb., and his height appeared normal. Visible gastric peristalsis was easily elicited by giving a drink of cold water. The tongue was slightly coated but there was no real evidence of chronic gastritis. The bowels were regular. The descending colon could be felt and appeared of normal size.

X-RAY EXAMINATION (H.C.G.). The stomach was much enlarged. Peristalsis was vigorous and indicative of hypertrophy of the gastric wall. More than 50% of the meal remained in the stomach at the end of 3½ hours. The duodenal cap appeared normal. The colon was distended and perhaps large (Fig. 5).

DIAGNOSIS. The presence of an enlarged and hypertrophied stomach is clearly proved clinically and radiologically in this case. As before, there is no evidence to show where the obstruction occurs, and it is suggested that it is due to chronic duodenal ileus.

COURSE. As the boy showed no symptoms and was making good progress he was discharged on June 12th without treatment.

Case 6. A.A., male, aged 6 years. First seen November 15th, 1929, on account of attacks of bronchial asthma which have recurred since he had broncho-pneumonia at the age of 8 months. He walked at 18 months. Tonsils and adenoids removed at 2 years.

On examination the upper abdomen was seen to be protuberant, the outline of the stomach was clearly visible, its wall could be easily palpated, and well-marked waves of gastric peristalsis were passing across the upper abdomen. After this observation the following facts were elicited :

The boy's appetite had always been rather poor, and had been worse since the change on to solid food at 18 months. His appetite had now become very bad and for the last two years there had been a loss of weight. There were periods of refusal of food and nausea, but vomiting was only occasional. When it occurred the vomiting was projectile. The boy stated that he felt very full after his meals and had a lot of flatulence. Hiccup occurred daily after food and was very persistent. About an hour after meals there was much gurgling in the stomach. The bowels tended to be loose rather than constipated.

The boy was admitted the same day to Paddington Green Children's Hospital. His weight was 43 lb., and his height appeared normal. His body was very thin but his muscles good. The chest showed some catarrhal sounds. The sputum and fæces contained no tubercle bacilli. On an ordinary diet gastric peristalsis of a powerful type was visible on many occasions, and the stomach felt hypertrophied. Splashing was persistently present. His appetite was not good but there was no real refusal of food. The stool on ordinary diet showed that the food was well digested : fæcal fat = 22% of dried fæces.

X-RAY EXAMINATION (H.C.G.) (November 21st.) Stomach was only a little larger than normal. Peristalsis was very vigorous soon after the ingestion of the meal, but later failed. There was much gastric delay showing a large residue in the stomach at 4¾ hours. No view of the duodenal cap was obtained. The condition found indicates one of mild obstruction with gastric hypertrophy and hyper-peristalsis (Fig. 6).

DIAGNOSIS. Mild obstruction probably due to chronic duodenal ileus.

COURSE. The boy was given a diet consisting of pounded and minced foods taken as dry as possible, with long intervals between meals. Drinks were taken in between meals. The foot of the bed was kept raised. On this régime the stomach became less obvious and the gastric peristalsis was much less in evidence. A small amount of weight was lost. He was discharged on December 11th, 1929.

Case 7. E.McC., male, aged 8 years. First seen January 6th, 1926. This was the first case seen by the writer showing visible gastric peristalsis in childhood. He was brought up for poor digestion.

He had always been a very poor feeder. In infancy he was seen by a pædiatrician who kindly reported that there had been no question of hypertrophic pyloric stenosis. Of late his appetite had been improving. He had had no abdominal pain and was occasionally sick. Hiccup was very persistent. The abdomen had been enlarged from the age of 18 months to 6 years. The bowels were rather constipated, but about every 2 months the stools became large, pale, and loose.

The boy was 49 in. in height, and was pale and thin. His eyes were puffy. The upper abdomen was enlarged and visible peristaltic waves passed from under the left ribs downwards and to the right. Splashing was elicited, proving the enlarged viscus to be stomach. The urine was normal : no indican, some oxalate crystals. Stool contained 20% of fat.

X-RAY EXAMINATION. (January 13th, 1926.) The stomach was very large hiding the duodenal cap and emptying slowly. Appendix retained barium over 72 hours (Fig. 7).

DIAGNOSIS. It was recognized at the time that there was some obstruction to the outlet of the stomach which did not show in the X-ray examination. It was thought that it might be a case of kinking of an atonic stomach. It is now suggested that the obstruction was due to chronic duodenal ileus.

COURSE. Dry meals with drinks in between were ordered. Massage daily to the stomach was given and a mixture of listerine, acid and nux vomica. Seen two months later the improvement in the boy's colour and appetite was satisfactory. He had gained ½-lb. in weight. The outline of the stomach under the abdominal wall could still be seen, but no visible peristalsis could be produced.

In April, 1929, the boy, now aged 11 years, was reported as very well, eating ordinary school diet, and weighing 66½ lb. in clothes. His appendix had recently been removed without incident.

Case 8. J.G., male, aged 12 years. Referred by Mr. Douglas Robertson for misshapen abdomen which it was thought might prevent his entrance into the Navy. First seen July 31, 1929.

The early history of this case was not obtainable. His abdomen was certainly enlarged at the age of 5 years when he had a severe attack of pneumonia. At that time the suspicion of fluid in the abdomen was raised, but no operation performed. Since then he had been in fair health but had suffered a good deal from attacks of vomiting. These now occur about four times a year: the vomitus will contain bile. He is at boarding-school and his general physique is improving, especially since he started regular drilling exercises. His tonsils and adenoids were removed without incident one year ago. The bowels are regular.

On examination the enlargement of the abdomen was all in the uppermost part where the stomach could be seen much distended and giving the abdomen a peculiar outline. Splashing could be obtained but no peristalsis was seen, nor did it appear after a draught of soda water.

X-RAY EXAMINATION (H.C.G.) On August 3rd, 1929, a 12 oz. opaque meal was given. The stomach was definitely larger than the normal for the patient's size. Peristalsis was normal and the emptying time average. The bulk of the meal had left the stomach in 2 hours. There was no evidence of any abnormality of the stomach except its size, and the duodenal cap was normal. Nothing was found to suggest the cause of the gastromegaly. There was no ileal stasis. The colon was voluminous and the transverse portion long and dipped low in the pelvis when the boy was erect. There was no evidence of any abnormal fixation of any part of the colon. At 24 hours the right half of the colon was emptying and a small quantity of the meal had been passed. There was, however, a definite tendency to stasis, there being a considerable residue at 24 hours. The bowels acted once daily during the examination: no aperient was used (Fig. 8).

DIAGNOSIS. The evidence of obstruction in this case is not conclusive, but it seems probable that hypertrophy of the gastric wall is present in view of two facts, first, the way in which the stomach could be seen standing up under the abdominal wall, and secondly, the normal emptying time of the very much enlarged stomach. As to the cause of the obstruction it is impossible to incriminate the attack of pneumonia or to suppose the presence of peritonitis at that time. Consequently it is suggested that the case is one of chronic duodenal ileus in which possibly the voluminous colon plays a added part in the production of obstructive symptoms.

COURSE. Progress being satisfactory, dietetic treatment and the continuance of drill were advised.

Case 9. D.N., female, born May 1st, 1923. Referred by Dr. R. K. White of Southsea for enlarged abdomen. First seen September 2nd, 1925, at the age of 2 years and 4 months.

The abdomen had been getting large for quite a year. In the mornings it measured 21½ in. in circumference, and at night, in spite of an action of the bowels at 6 p.m., it was usually 25 in. There was much gas in the stomach and intestines. The motions were inclined to be alternately constipated or loose: they were always of good colour. She appeared to feel quite well, and had a high colour. She eat hungrily the rather restricted diet permitted. She was not thin and was growing steadily in height.

On examination the abdomen was seen to be much distended and tympanitic all over. Megacolon was suspected but no peristalsis of stomach or colon was observed. Urine contained an excess of indican. The fæces showed no excess of fat on a high-protein, low-fat diet: muscle fibres were not well digested.

Seen again in February, 1926, there had been general improvement following a course of colon lavage. On a more normal diet the fæces showed 37·2% of fat.

FIRST X-RAY EXAMINATION. (February 23rd, 1926.) The stomach showed great enlargement containing a great excess of gas. Its emptying time was slightly over the normal. The duodenal cap was not seen. The colon also showed great dilatation, which was confirmed by an opaque enema.

DIAGNOSIS. The enlargement of the colon was taken to be atonic distension secondary to fat decomposition comparable to that noted by the present writer in a case of cœliac disease,

The condition of the stomach was regarded as in some way not understood secondary to the megacolon (Fig. 9).

COURSE. From this time the girl was kept on a low-fat diet and gradually improved. She kept a good colour, had very pink cheeks and gained in weight and height, and eat well. She was a matter-of-fact little girl, who liked her doctors and enjoyed her X-ray examinations, but without any morbid self-interest. All these points were against a diagnosis of cœliac disease, but on the other hand any extra fat in the diet produced abdominal distension which otherwise was not too great.

SECOND X-RAY EXAMINATION. (March 1928). The stomach and colon were enlarged as before, but their tone was improved. The gastric peristalsis suggested the presence of hyper trophy of the wall of the organ.

On May 29th, 1929, the girl was seen again and the stomach was distended by a draught of soda water. Well-marked peristalsis was seen passing across the abdomen in the gastric region. She was now aged 6 years, weighed 47¾ lb., and measured 44½ in. in height. Since July 9th, 1926, she had gained 16¼ lb. in weight and grown 7½ in.

DIAGNOSIS. It is suggested that the obstruction to the stomach thus tardily recognized is due to chronic duodenal ileus, and that the dilatation of the colon is a factor in the production of the obstruction. The cause of the enlargement of the colon is obscure: it may be due to fat indigestion secondary to the obstruction to the bile in the duodenum. It is unlikely to be the result of cœliac disease of the ordinary type.

APPENDIX B.

Recorded cases of chronic duodenal ileus in children.

Bernheim-Karrer[28] (1904). Boy aged 8 months at death. Soon after birth he began to vomit breast milk. The vomiting persisted in spite of treatment. Obstinate constipation was also present. He developed frequent convulsions and ultimately died suddenly in his bath. The convulsions were regarded as due to toxic products of decomposition in the stomach and duodenum. During life no examination for visible gastric peristalsis was made.

At autopsy the superior mesenteric artery was found to be compressing the duodenum, and the duodenum above the constriction was so much dilated that it resembled a second smaller stomach.

The author refers to Albrecht's[7] paper (1899) on duodenal ileus in adults, and states that the case recorded is the first one reported in a child.

Frank[29] (1913). CASE 1. Girl, first child. Breast-fed for 4 months. Gained in weight until the age of 4½ months when vomiting started, at first immediately after food, but later once only in the day, independent of food. The vomits became very large and seemed to consist of more than the last feed. Sometimes they were green, but never black. Increasing constipation was present. The mother had noticed excess of gurgling in the abdomen but no enlargement.

When first seen (August, 1911) at the age of 11 months, the child was very much wasted, weighing 4,650 grm. The abdomen was strikingly enlarged in its upper half, and left-to-right peristalsis was clearly seen in the epigastric region. The enlarged stomach could be seen and felt, and to the right of it was a smaller swelling. These could be emptied manually and gave the impression of emptying through a constriction. On passing a stomach tube much air escaped and the epigastric fullness collapsed, the circumference of the abdomen decreasing from 43 cm. to 38 cm. The stomach contents contained bile, free HCl, but no blood. The child took food hungrily but was restless after feeding until a large vomit brought relief. Loud borborygmi were noticed.

After 10 days treatment without progress the abdomen was opened. There was found great distension of the stomach and duodenum up to the root of the mesentery: on the other side of the mesenteric root the bowel was collapsed. Adhesions were present round the gall-bladder and duodenum. After dividing the ligament of Treitz, duodeno-jejunostomy was performed.

As a result of the operation the vomiting stopped and the child's weight increased regularly; 18 months later the girl was said to be well-developed and strong, with a normal digestion.

The author in discussing this case regarded it as an example of duodenal ileus in a child.

Case 2. Boy. Healthy during the first year of life and then lost appetite and began to vomit. Vomits were very large, '2 meals at a time.' Vomiting stopped after 14 days for several weeks during which time there was a slight increase in weight. The sickness then returned but was followed by another period of quiescence. The circumference of the abdomen was increased.

Boy was first seen (August, 1912,) at the age of 2 years. He was very wasted: weight = 7,300 grm. The upper abdomen was very prominent, and by blowing up the stomach the prominence could be proved to be due to an enlarged stomach. The greater curvature reached down to within 2 finger-breadths of the umbilicus. Further distention could be made out in the region of the duodenum. X-ray examination showed two shadows, the stomach and duodenum, divided by a broad lighter part corresponding to the pylorus. After 1½ hours none of the meal had passed out of the duodenum. After 4½ hours a fairly large amount had passed into the bowel, but at 8 hours the duodenum was still well filled. A diagnosis of chronic occlusion due to arterio-mesenteric ileus was made.

Seen 6 months later there had been no further vomiting, and the physical signs remained as before. Operation was recommended and refused.

The author notes that the apparent absence of bile in the vomit in this case is probably to be explained by the loss of colour through dilution.

Downes[30] (1917). Boy, weight at birth 6 lb. Breast-fed for 5 months. Vomiting started almost from birth and got gradually larger in volume. Vomit contained food taken two or three days previously. The sickness would stop at times for periods of one or two months. Attacks of diarrhœa occurred about every two months. They were accompanied by much prostration and the passage of blood and mucus in the fæces.

When first seen at the age of 4½ years, the boy weighed 29 lb. in his clothes. Irregular gastric peristalsis could be seen, and definite duodenal peristalsis, passing from above downwards below the region of the pylorus was also seen. X-ray examination showed the duodenum to be almost as big as the stomach.

At operation the wall of the stomach was found to be thickened. The pylorus easily admitted the tip of the index finger. The duodenum was dilated to almost the size of the stomach and its wall was three or four times its normal thickness. The distension involved the whole of the duodenum, ending abruptly where the gut passed under the superior mesenteric artery. A posterior no-loop gastro-enterostomy was performed and the pylorus closed with silk ligatures. The child did well until the pylorus reopened. Later the pylorus was divided in a second operation and the improvement afterwards was satisfactory.

DuBose[31] (1919). Female, mongol: weight at birth = 7½ lb. Began vomiting yellow fluid on the third day. At 2 months was seen by author for vomiting. Weight then = 5½ lb. Pyloric stenosis was excluded on account of the presence of bile in the vomit, the absence of palpable tumour, and the X-ray examination which showed megaduodenum. On opening the abdomen it was observed that the duodenum was of enormous size, dilated and flaccid. There was neither diverticulum, constriction, adhesive band, nor kink of the duodenum, but the jejunum was collapsed beyond its origin at the mesentery, having an appearance such as is found in mechanical obstruction of the intestine caudal to the point of obstruction. 'It is probable that in this case we have a true arterio-mesenteric obstruction at the duodeno-jejunal flexure of congenital origin . . . rather than a congenital giant duodenum.'

Jewesbury[32] (1922). Male, aged 9 days. From the 3rd day of life projectile vomiting and wasting developed. Slight jaundice was present. Gastric peristalsis was visible. Feeds gave rise to discomfort: vomits were large, forcible and bile-stained.

At autopsy the stomach and duodenum were enormously dilated and hypertrophied. Below the duodeno-jejunal junction the bowel was very shrunken and small. The cæcum was abnormally high and under the liver. The obstruction in the duodenum was caused by pressure from outside by surrounding structures, and in particular the right colic artery.

Henske and Best[33] (1928). Child weighing at birth 8 lb. 1 oz. Vomiting started from birth and was accompanied by loose motions. At 5 months its weight was 11 lb. At this time

a diagnosis of pyloric stenosis was made. An X-ray showed gastric stasis and dilated duodenum, but the latter 'was overlooked at the time.' Under treatment the child improved and at 12 months weighed 21 lb. At 13 months there was fresh vomiting, the vomit being green fluid mixed with food. The bowels were again loose. Recovery ensued, but at 21 months the baby died in another similar attack.

At autopsy the cæcum was found in the middle line just behind the umbilicus, and the large intestine was not anchored. 'There was definite partial obstruction at the duodeno-jejunal angle, caused not only by the twisting and fixation of the jejunum on its longitudinal axis . . . but also by the dragging of the transverse colon. The mesenteric root was also compressing the jejunum. Naturally, the loaded ascending and transverse colon increased the obstruction more than the empty colon, and permitted attacks of duodenal toxicosis, which resulted in vomiting and dehydration.'

Quervain [39] (1907). Girl, aged 13 years, the subject of disease of the cervical spine and compensatory lordosis in the lumbar region. The stomach and the duodenum, which, as operation showed, reached considerably to the right, were greatly distended, and gave evidence of splashing on auscultation. The stomach was tympanitic and the duodenum yielded a metallic note. The cervical disease prevented the use of the knee-elbow position for the purpose of treatment, and the fact of tuberculosis did not, of course, exclude obstruction by a band. Laparotomy was, therefore, performed and showed the whole of the small intestine deeply in the true pelvis. The kink was situated at the junction of the duodenum with the jejunum. The latter became filled with gas as soon as it was lifted up.

Quain[38] (1920). Girl aged 17 years. Stated to have been quite well until she was 8 years old. She then started to have pain in the stomach immediately after food and lasting ½ to 1 hour. Often this was associated with the vomiting of bile which give partial relief for a day. After about 4 years the pain and vomiting were less constant but would occur at least once a week. Her appendix was removed at 14 without relief. For 3 months before coming under the care of the author the pain had been much worse, occurring after each meal and before breakfast. Bile was vomited almost daily. Severe constipation was present: laxatives led to increase in the pain. The girl complained of almost constant dull headache.

At 17 the girl was small, thin and pale. X-ray examination showed enlarged stomach with ptosis nearly to the pubes. Delay in the second part of the duodenum was observed. The gastric juice was practically normal.

At operation a membrane running from the descending duodenum to the liver at the right of the gall-bladder was found. No glands were present. The third part of the duodenum was much dilated and formed a 'U' with its angle reaching to nearly the pelvic brim, and its distal end passing upwards to the superior mesenteric artery. This ascending part was over 2 inches in diameter and showed thickening of its wall. The dilatation of the gut ended abruptly at the superior mesenteric artery in an abnormally narrow jejunum. 'It was clear that the constriction had been chronic enough to cause both dilatation and hypertrophy of the duodenum.

Duodeno-jejunostomy was performed and gave complete relief to the patient. She gained 30 lb. in weight in 3 years.

Higgins[19] (1926). Boy, aged 7 years, had had for several months epigastric pain which at times was severe. Vomiting occurred in attacks. Constipation was present. X-ray examination revealed an incomplete obstruction at approximately the point where the superior mesenteric artery crossed the spine. Above this point the duodenum was dilated and reverse peristalsis was present. A diagnosis of dilated duodenum was made. Improvement occurred under treatment by means of a special diet and the use of an abdominal binder.

Wilkie[22] (1027). In a series of 75 cases of chronic duodenal ileus the youngest patient was a boy aged 7 years. The only account of this case is as follows:—The most marked case of duodenal ileus with which I have met was that of a boy aged 7 years, in whom, owing to an abnormal rotation of the bowel, the duodenum was twisted sharply around the mesenteric vessels with resultant recurring attacks of complete duodenal obstruction which brought him frequently to the point of death. These attacks were finally stopped by short-circuiting the bowel around the difficulty.

Ombrédanne[40] (1919). Girl aged 12 years who had suffered since she was 18 months old from bilious vomiting occurring two or three times a month. Gastric peristalsis was observed and a duodenal tumour detected. Tetany was present. At operation there were found arterio-mesenteric compression together with volvulus. The volvulus was put right and colopexy performed.

Wheelon[41] (1921). Baby of 7 months who had had ' gastric disturbance ' since birth. For one week had had bilious vomiting, constipation and emaciation. X-ray examination showed megaduodenum, suggesting kinking of the small intestine about the region of the ligament of Treitz, or at the point of emergence of the duodenum from its retro-peritoneal position. Operation discovered a mass in the left side of the pelvis which showed small intestine in a sac of peritoneum, and had slipped through an opening in the root of the mesentery, thought to be congenital. The mass was reduced, and the mesenteric slit sewn over. The child died.

During life no visible gastric peristalsis was noted, but waves of peristalsis were seen which were taken to indicate obstruction of the terminal portion of the duodenum or upper part of the small bowel.

Foucar[42] (1923). Boy aged 4 years. Suffered from pulmonary fibrosis and bronchiectasis resulting from whooping-cough. Since 1 week old he had been subject to attacks of vomiting three or four times a year, each attack lasting two or three weeks. During them he vomited 2 to 10 times a day without relationship to meals. The vomit was copious in amount and projectile in character, at first containing some food but later greenish mucoid material only. With the attacks were some fever, and pain in the umbilical region. He was admitted to hospital for a plastic operation on the chest.

At 4 years he weighed 27 lb. Under observation the outline of the enlarged stomach was seen extending below the level of the umbilicus, and definite gastric peristalsis was seen. An X-ray examination made during a quiescent period showed no evidence of obstruction in the stomach or duodenum : the colon was somewhat dilated. Later, during an attack of vomiting, the examination was repeated and then revealed obstruction to the duodenum about 15 cm. from the pylorus.

At operation the duodenum was found dilated as far as the upper jejunum. The small intestines were matted together by dense adhesions and had become twisted. This volvulus, through the bands of adhesions, closed the lumen of the duodenum also. The volvulus was unfolded and the adhesions freed. Six months later the child was reported as well and having gained much weight. ' It was concluded that the child was suffering from adhesive fœtal peritonitis, resulting in a volvulus of the small intestines which closed the lumen of the duodenum secondarily, resulting in intermittent attacks of obstruction. When the volvulus unfolded itself, the symptoms ceased, only to return when the mass of small intestines became twisted on itself, and on account of adhesions, closed the duodenum also.

Waugh[43] (1928). Boy, aged 7½ years. There was a history of severe pain and vomiting since infancy. At operation a cysticocolic membrane passing over the second part of the duodenum to end on the hepatic flexure of the colon caused tight constriction of the duodenum. In addition a tendency towards arterio-mesenteric ileus was found. The band was divided and the ileus was left untreated. Although it was felt that possibly further treatment might be necessary for this, the patient did well without a second operation.

Judd and White[44] (1929). Case 1. Male, aged 19 years. Was well until 2 years of age, when attacks of generalized abdominal pain began, recurring every 2 or 3 weeks. Pain occurred very soon after food, and was frequently associated with vomiting, from a few minutes to several hours after food, which always afforded relief. Appendix found on the left side and was removed without relief. X-ray examination of the stomach was negative. Operation revealed ' chronic intestinal obstruction due to congenital malformation of the small intestine apparently at the duodeno-jejunal angle. The duodenum was dilated to about three times normal size ; the distal half was completely covered with peritoneum and possessed a short mesentery. There was a very firm band 7·5 cm. wide extending from the root of the mesentery across the duodeno-jejunal flexure and becoming continuous with the posterior leaf of the transverse mesocolon. This band held the bowel fixed at this point and produced partial occlusion. The root of the mesentery, instead of having its normal attachment to the posterior abdominal wall . . . seemed

to be attached to a limited area anterior to the 2nd lumbar vertebra, being pedunculated, as it were. . . . The cæcum and ascending colon possessed an abnormally long mesentery, thus allowing for motion of that part of the bowel. The peritoneal band was separated, freeing parts, but not releasing the obstruction entirely. Therefore a lateral anastomosis was made between the duodenum and the loop of jejunum just below the point of obstruction.' Patient reported well 12 months later.

Case 2. Female, age 19 years. Since she could remember she had had attacks of pain in the umbilical region, worse after meals, relieved by vomiting, and lasting 7 to 10 days. Up to 17 these recurred about twice a year; since then every 2 months. X-ray of the stomach and colon was negative. Operation revealed the duodenum 'distended and hypertrophied: it did not rotate up behind the stomach but came directly and obliquely downward and was compressed over the vertebræ by the weight of the mesentery of the small intestine. Instead of presenting on the left side of the vertebræ, the jejunum began in front and on the right side of the vertebræ. The peritoneal fold was divided in order to give a larger opening as the retroperitoneal portion of the duodenum became the jejunum. It was felt that the liberation of the constricting bands was sufficient to relieve the partial obstruction, though duodeno-jejunostomy might be necessary later. The immediate post-operative convalescence was uneventful, but the ultimate results were only fairly satisfactory.'

Foucar[42] (1923). Boy, aged 18 months. Weight at birth = 8 lb. He had suffered from attacks of vomiting since birth except from the 3rd to the 5th month. The vomiting occurred 1½ to 2 hours after food and was projectile. Vomitus consisted of greenish yellow fluid containing very little food, but occasionally food given 6 hours previously would be brought up. For 12 months the child's weight had been stationary, attacks of vomiting occurring every week, and lasting one or two days. For the past 8 months the parents had noticed wave-like movements crossing the abdomen during the attacks. The presence of gastric peristalsis on the days of vomiting was confirmed by the author. An X-ray examination, made during a quiescent interval, did not reveal any obstruction. At operation 'the mesentery of the small intestine was found to be markedly œdematous, but the pylorus appeared normal. The thickening of the mesentery was due to marked enlargement of the mesenteric glands. Neither bands of adhesions nor obstruction were found. The appendix was removed and also a mesenteric gland . . . . The mechanism of this case would seem to be transitory obstruction due to inability of the intestines to adjust themselves because of the thickened œdematous mesentery.'

GASTROMEGALY FROM CHRONIC DUODENAL ULCER IN A CHILD

BY

REGINALD MILLER, M.D., F.R.C.P.

Physician, Paddington Green Children's Hospital, London.

The single case recorded here falls naturally into three chapters. (1) A previously healthy female baby began at the age of six months to suffer from vomiting which continued more or less up to the age of seven years. (2) At seven she showed extreme gastromegaly from obstruction to the evacuation of the stomach which operation discovered to be due to a chronic cicatrizing duodenal ulcer. Gastro-jejunostomy gave permanent relief from the symptoms of ulcer. (3) At the age of 28 her abdomen was opened for gall-stones and the scar of the old ulcer adherent to the head of the pancreas was seen.

Case report.

A.W., female, born 1900. Admitted on February 20th, 1907, aged 7 years, to the Paddington Green Children's Hospital, under the care of Dr. G. A. Sutherland, for vomiting, wasting and thirst.

The history was to the effect that the baby had been quite well until weaned at the age of six months. Vomiting had then started and had been very troublesome for six months. It had then subsided to some extent but had never entirely stopped. She would vomit once or twice a week, and the vomit would be projectile in type, 'coming all of a spurt as if she had been overloaded.' The vomiting was sufficient to retard progress considerably.

For 18 months before admission the vomiting had been more frequent and had led to the loss of much weight. It had occurred chiefly in the evening, especially on going to bed for the night, when all the food of the day would be brought up. The appetite had become extremely bad, but after being sick she would often ask for food. There had been no pain except immediately before the act of vomiting. There had been great thirst. The bowels had been usually constipated, but occasionally relaxed. At no time had blood been noted in the vomit or stools.

On admission, aged 7, she was very wasted and wizened, although not stunted in height. Her weight was only 27½ lb. She was frequently sick, and the vomits were large and projected with great force. On one occasion she vomited 52 oz. The vomit was frothy and contained mucus and fragmentary food. Gastric peristalsis was persistently visible in the three large waves typical of hypertrophy of the muscle of the stomach. No tumour could be felt, but splashing in the stomach was easily elicited. The bowels were constipated.

A course of gastric lavage, which brought away much mucus, combined with the use of small feeds, caused some improvement in the appetite, but there was a loss of weight of 2 lb. in a month.

On March 18th, 1907, Mr. F. F. Burghard operated and wrote the following note :—'Gastro-jejunostomy performed by posterior no-loop method. Much thickening of the first part of the duodenum, chiefly on the posterior aspect, but extending round five-sixths of the circumference near the pylorus. Many fine adhesions about the pylorus, the peritoneal coat of which was very vascular.'

By March 30th the child was on full diet, and she was discharged on April 17th, having gained 6 lb. weight since the operation.

DIAGNOSIS: duodenal stenosis from cicatrizing duodenal ulcer dating from infancy.

AFTER-HISTORY. In May, 1929, I was able to get into touch with her. She was then aged 29 years, was 5 feet in height, and well nourished. She stated that after the operation in 1907 she had kept free of any abdominal symptoms until after she had turned 21. She had then begun to suffer from flatulence and some pain after her meals, and these symptoms had gradually increased although at no time had she been sick. In August, 1928, she had had a severe attack of gall-stone colic with jaundice, and in the following month she had been admitted on this account to the West London Hospital, to the Medical Superintendent of which hospital I am indebted for the following facts :—

X-ray examination of the alimentary tract showed that the old gastro-jejunostomy was functioning well. At operation an old ulcer in the region of the pylorus was found adherent to the head of the pancreas. The gall-bladder contained numerous small stones, and was removed. A pathological section of the wall of the gall-bladder showed inflammatory changes throughout. The patient was discharged in October, 1928.

In May, 1929, she stated that since the cholecystectomy the abdominal symptoms had disappeared. She still had the sensation which was habitual with her, that any large meal overfilled her, but this could easily be obviated by taking small meals at frequent intervals.

Discussion.

At each of the three periods of this case there are points of interest to be discussed. (1) Although there is now a considerable literature on the subject of infantile duodenal ulcer, it most of it dates, as Holt[1] has observed, from 1908. The present case showed the symptoms of ulcer in 1900 and gastro-jejunostomy for the relief of the stenosis due to cicatrization was performed in 1907, a comparatively early date for this operation. We have therefore in this case, considered either as one of infantile ulcer or of gastro-jejunostomy, an exceptionally long after-history.

The first symptoms of duodenal ulcer did not appear until the sixth month of life. This is rather later than the majority of cases of this type. Palmer[2], for instance, in 65 cases found that only 5 were over the age of five months. Adriance[3] has recorded an example in a child of ten months. The onset of the symptoms occurred at the time of weaning, but this is probably no more than a coincidence, as it is not the rule to be able to trace any dietetic factor in the production of duodenal ulcer in infancy. There has been some discussion (Helmholz[4], Torday[5]) whether atrophic conditions of infancy predispose towards ulcer, but it seems certain that, as in this case, ulcer may develop suddenly in previously healthy babies. Ulcer of the duodenum has also been described (for instance, by Bloch[6]) as a complication of hypertrophic pyloric stenosis. The ulcer in infancy is usually immediately distal to the pylorus, as in the present case.

The projectile character of the vomiting, as seen in this case from the start, has been noted by Holt and Palmer. It is presumably due to the spasm of the pylorus set up by the ulcer in its immediate neighbourhood. Visible gastric peristalsis and a palpable pyloric tumour are rare (Palmer). Although the vomiting in this case persisted for so long, no gross bleeding was noted at any time. This is in accord with Palmer's opinion that hæmatemesis and melæna are rarely seen in infantile cases 'until the end of the scene,' death usually occurring within 48 hours of such bleeding.

The most unusual feature of the present case is its chronic course. The vomiting which started at the age of six months, never really subsided until the child was operated on at the age of seven years for duodenal stenosis. It is not easy to say at what age the cicatrization of the ulcer led to stenosis of the duodenum, but clearly from the age of 5½ years onwards the vomiting was due to the stenosis. This led to extreme gastromegaly and emaciation.

(2) At operation at the age of 7 years, the present case showed a chronic duodenal ulcer which had given rise to symptoms since the age of six months. Duodenal ulcers in children past the age of infancy are most commonly of tuberculous origin, as in the cases recorded by Gerdine and Helmholz[7], and as such are of little clinical interest.

A chronic peptic ulcer in a child is certainly extremely rare. Proctor[8] in 1925 reviewed the whole subject. He found that out of 6,664 cases of duodenal ulcer at the Mayo Clinic only one was in a child, which case he recorded ; but he noted that in 1,000 cases of duodenal ulcer in adults no less than 26 gave a history of symptoms dating back to childhood 'even to the age of four or five years.' Searching the literature for recorded cases of chronic peptic ulcer in children, Proctor was only able to collect 19 satisfactory and 2 questionable examples ; and of these, three were instances of chronic duodenal ulcer (Bichat[9], Alsberg[10], and Palmer[2]). To these Proctor added the one instance from the Mayo Clinic already mentioned. Since Proctor's paper some other examples of duodenal ulcer in children over the age of infancy have come to light, although the chronicity of some of them is perhaps open to question. The list of these cases so far as I have been able to trace them is as follows :—

Bichat[9] (1910). Girl of 14 years showed perforated duodenal ulcer. Symptoms for 4 years previously. Recovery by operation.

Alsberg[10] (1920). Girl of 2 years with sudden pain and bleeding. Operation showed duodenal ulcer scar near pylorus with adhesions to the liver. Recovered.

Palmer[2] (1921). Boy of 6 months who had had symptoms almost from birth. Pyloroplasty followed 18 days later by gastro-jejunostomy. Recovered.

Proctor[8] (1925). Boy of 13 years : had had symptoms for 4 years. Old ulcer of duodenum extending round pylorus with great thickening and contraction. Gastro-enterostomy. 8 years later reported that he had kept quite well.

Carrick[11] (1924). Boy of 10½ years. Symptoms for 2 years. Gastromegaly, visible peristalsis, stasis. X-ray diagnosis.

Dickey[12] (1926). Three cases diagnosed by skiagram, no operations. All boys. (1) Aged 9, with a history of pain 'ever since he could remember.' (2) Aged 11½, with attacks of pain since 5 and melæna at 5 and 8 years. (3) Aged 8 years, with symptoms for 4 months.

Dickey[13] (1926). Boy of 13 years with intermittent epigastric pain for 1 year. Operation showed ulcer and diverticulum, both in the first part of the duodenum.

Paus[14] (1926). Boy aged 14 years who had had the disease 'as long as he could remember.'

Pedrazzi[15] (1927). Case diagnosed by skiagram.

Pototschnig[16] (1927). Perforated duodenal ulcer, not of infantile origin, in a child of 11 years.

Flint[17] (1927). Boy aged 12 years showed at operation a large mass surrounding a duodenal ulcer.

These cases indicate that should a child suffer from a chronic duodenal ulcer it will probably have acquired it after the age of infancy. The present

case, showing a chronic duodenal ulcer at seven years with a history dating back to the sixth month of life, seems in this particular to be unique. It may therefore be suggested that in such cases of infantile duodenal ulcer as survive healing is usually successful and complete.

The same point is brought out by the absence of scars in the duodenum in children. Gruber[18], who examined with extraordinary care the duodenum in all autopsies he made for the five years 1906 to 1910, found in the age group 1—10 years ulcers in 10 cases, erosions in 7 cases, but no cicatrices. These last were not found at any age under the decade 30 to 40 years. Palmer also has noted how quickly and successfully healing takes place in the duodenal ulcer of infancy.

(3) No particular comment is necessary to display the interest in the lengthy after-history in the present case. This must be unique amongst recorded cases of infantile duodenal ulcer.

Summary and conclusions.

1. A case of duodenal ulcer arising at the age of six months is followed until the patient attained the age of 29 years.

2. Chronic duodenal ulcer is extremely rare in children of any age. Where it is found in older children it has not usually arisen from an ulcer originating in infancy. Similarly, scarring of the duodenum is of great rarity in children.

3. It is concluded that if an infant with duodenal ulcer survives the acute early period of the disease, healing of the ulcer is likely to be complete.

I am indebted to my friend, Dr. G. A. Sutherland, for his kind permission to make use of the notes of his case reported here.

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GASTRIC SECRETION IN INFANTS AND CHILDREN

BY

A. V. NEALE, M.D., M.R.C.P.

(From the Children's Hospital, Birmingham.)

A simple, rapid and satisfactory method of investigating the gastric secretion in infants and children is of undoubted value in their clinical investigation. The difficulties experienced by the use of the ordinary methods in children necessitates a new approach. With the usual method of passing a gastric tube followed by a test meal, the psychical and physical disturbances which ensue must have an adverse effect upon the functions in question. The swallowing of considerable amounts of saliva and mucus introduces difficulties. The gastric fluid removed after a test meal consists of an intimate mixture in which there is no pure gastric juice. The juice has undergone considerable change, such as dilution, buffering and partial neutralization. The variations in the type of meal used, both qualitatively and quantitatively, the frequency of vomiting during the test, and the length of time usually required for its completion all tend to lessen the value of the method.

Our aim therefore has been to elaborate a technique which eliminates swallowing a meal and at the same time causes no disturbance to the child. In order to dispense with the neutralizing and buffering effects of the milk meal, a dilute aqueous solution of ethyl alcohol has been used, following the suggestion of Chesney[1].

In all the experiments used for this investigation, the conditions were kept as constant as possible. The collections were made in the morning after a night's fasting, except in the case of infants where there was an interval of about six hours. A special tube was devised, consisting of a small narrow perforated light metal tip connected to very narrow rubber tubing, the width allowing the tube to be passed easily through the nose of even the smallest child. The nasal route has several particular advantages, causing much less general disturbance, no vomiting, and no increased salivation, because the soft rubber does not irritate the tongue or pharyngeal wall, and also allows the child to breathe easily and eliminates damage to the tube by the teeth. The metal tip aids in guiding the tube into the œsophagus and provides a means of moving the aspirating end around in the stomach in order to obtain all the fluid therein. The resting juice is completely aspirated with a syringe; 20 to 40 c.cm. of neutral 7 per cent. ethyl alcohol in distilled water at body temperature is introduced slowly into the stomach through the tube. The child is quite unconscious of the admission of the fluid and remains undisturbed. No swallowing occurs and salivation is at a minimum. At intervals of ten

or fifteen minutes over a period of one hour, 10 c.cm. are removed, the stomach being completely emptied at the final aspiration. Normally the fluid obtained is clear and watery.

Information obtainable from these specimens includes the presence or absence of suspended matter, and the acidity. No absolute values for the volumetric gastric secretion can be obtained by this method because there is no control of the volume of alcohol-water which has left the stomach either by absorption or passage onwards. However, in cases where only a knowledge of a qualitative acid secretion is desired the information is yielded. In a large series of infants and children no instance occurred where the alcohol-water failed to excite a flow of acid gastric juice, but in some cases the secretion apparently ceased after thirty minutes.

Owing to the confusion which has appeared in medical literature concerning the terms achlorhydria and achylia gastrica, it appeared imperative that in all cases where an apparent failure of acid gastric secretion occurred after the ordinary meal stimulus, or if anacidity was found, a convenient or separate test should be carried out by the use of histamine as detailed below, the gastric tube being left in situ.

Investigations with histamine.

This substance, a decarboxylation product of histidine, was introduced for use in man by Carnot in 1922, who observed an active secretion of acid gastric juice following injection. Since this date various investigators have used the substance in experimental studies on gastric secretion in adults; the work having been facilitated by the preparation of histamine in a pure state. Pharmacologically it is practically ineffective when given by mouth, but by subcutaneous injection it causes a transitory circulatory effect by capillary dilatation as seen by cutaneous flushing. With a dose sufficient to produce a maximal flow of gastric juice no adverse symptoms are produced. In a very large series of patients, the specially pure product of Imido-Roche of Paris has given excellent results. The stimulation of gastric secretion would appear to be a specific effect of the substance. Evidence has recently been obtained to show that no stimulation occurs in the case of the other digestive secretions.

Until the present investigation was carried out, histamine had been used on adults only. Polland[2] observed that variations in the height of chloride concentration in the gastric secretion after histamine represented individual differences. It is unknown whether chloride always leaves the secretory cells at a constant rate, but the juice as poured into the stomach shows differences in various people. The general character, however, of the curve of secretion volume is similar in all persons, there being a rapid rise in volume during thirty minutes immediately following histamine, and a continued fall thereafter until at sixty minutes the previous resting rate is regained. In pure specimens the curves of the titratable acidity and total chloride run practically parallel. The former may therefore be utilized as a fairly accurate measure of the chloride-secreting power of the stomach following histamine stimulation.

Polland's observations indicate that provided that pure gastric juice is examined, the titratable acidity at the height of secretion furnishes an index of the acid secreting power of the stomach glands, as it falls short of the actual chloride secretion by a constant amount. Careful observations show that all the gastric secreting functions show a co-ordinated response to histamine. Increase in titratable acidity results in a greater output of chloride than of base. Nitrogen is also actively secreted. The non-protein nitrogen secreted is proportional to the concentration in the blood, but like the total chloride content, it is always below the blood level.

Active secretion of peptic enzyme also follows the injection of histamine. Cassel[3] has adequately demonstrated the power of histamine juice to digest protein. This worker has successfully carried out experiments which show that the juice rapidly converts inactive beef-steak into an active form which stimulates the production of a reticulocyte response in pernicious anæmia. There appears to be little doubt, therefore, that the gastric secretion following histamine injection is physiological in nature and represents the maximal functional level of the gastric secretory power in the individual concerned. Histamine injection in pernicious anæmia fails to produce any acid gastric secretion. By this means the true achylia gastrica is confirmed. Many conditions have been described as associated with achylia gastrica. These ideas have been dispelled since the active functional power following histamine has been determined. Atropine has no inhibitory effect upon the gastric action of histamine. Bloomfield and Polland[4] have further shown that repeated examination in the same person after a similar dose of histamine produced secretion curves in almost complete agreement. It is obvious, therefore, that the method may be applied for the study of the secreting powers of the stomach in children provided an accurate knowledge of the suitable dose of histamine for the child's weight and age is known. Careful experimentation has furnished an optimum dosage for the different age-groups studied in this paper.

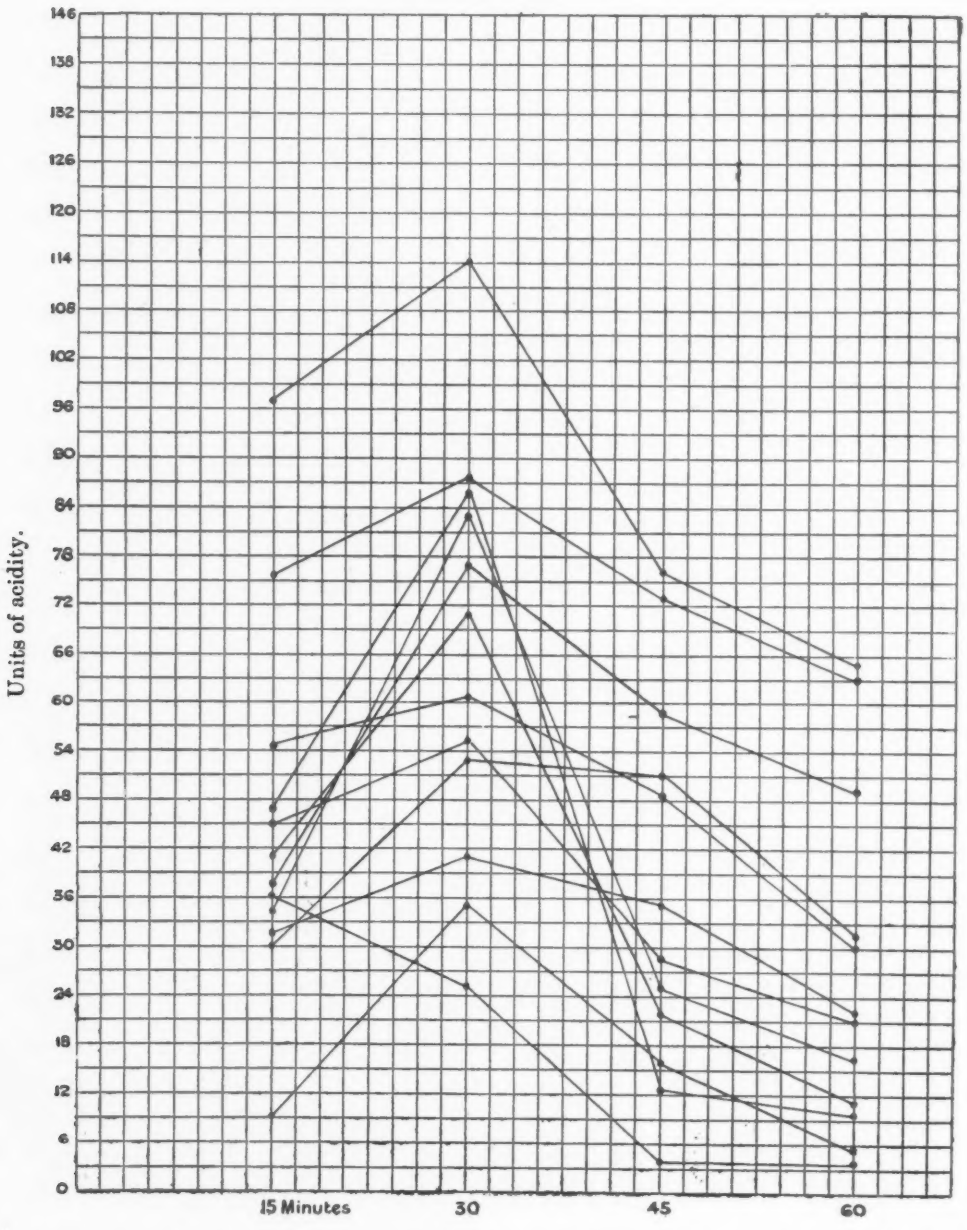
The object of the present investigation following the use of histamine was to determine if possible the volume and acidity of the pure gastric secretion occurring in infants and children. Obviously, by other methods, such as the use of test meals or even of alcohol-water mixture, this information has been impossible to obtain. By the use of histamine no diluent action or other disturbing factor is introduced. By experience of the action of histamine during duodenal drainage experiments it has been learned that the secretion of the stomach following injection remains within the stomach during the period under investigation :—namely, the sixty minutes immediately following. Hence volumetric estimation of the gastric secretion is rendered fairly accurate by the method.

Dosage.—The optimum quantity of histamine (Roche) to be used has been determined by a large number of experiments. Careful use of a range of doses in the same patient, in several instances, has shown that there is a certain optimum quantity of histamine which will produce a maximum acidic concentration in the individual. A greater dose will produce a larger volume

but the acidity remains approximately the same as with the smaller optimum dose. In the different age groups studied, fortunately the optimum dose has been found to coincide with the amount which produces signs of general

CHART A.

INFANTS AGED ½ TO 1½ YEARS. GASTRIC ACIDITY.

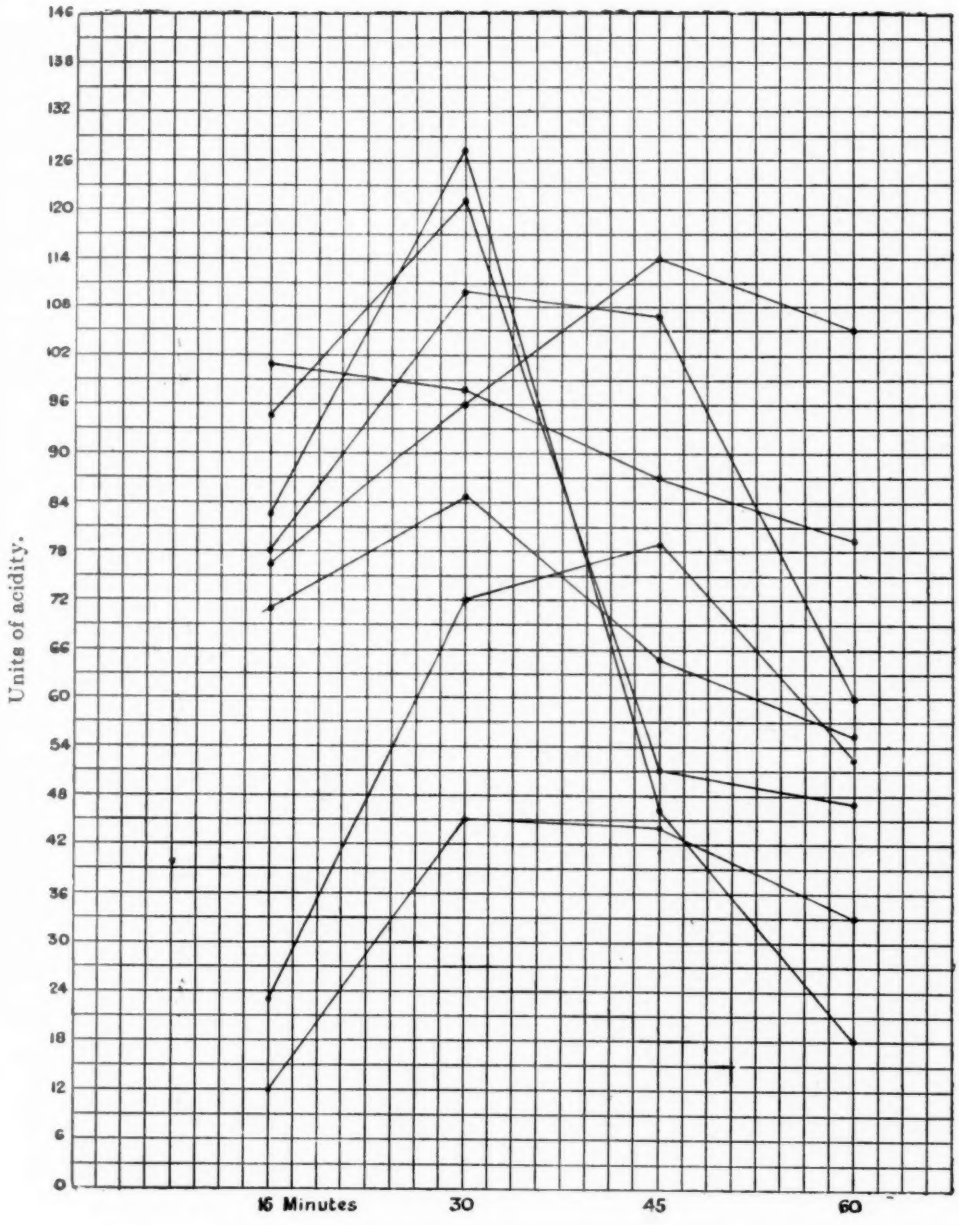


histamine effect without any adverse reactions. The reactive indications are a moderate generalized cutaneous hyperæmia and a slight acceleration of the pulse rate, without headache or signs of collapse. No rule could be adopted for calculation of the adequate dose according to the child's weight

but trial has shown that up to 2 years of age 0·15 mgrm., from 4 to 6 years 0·2 mgrm., and from 10 to 12 years 0·3 mgrm. have been correct for individuals within the average.

CHART B.

CHILDREN AGED 4 TO 6 YEARS. GASTRIC ACIDITY.

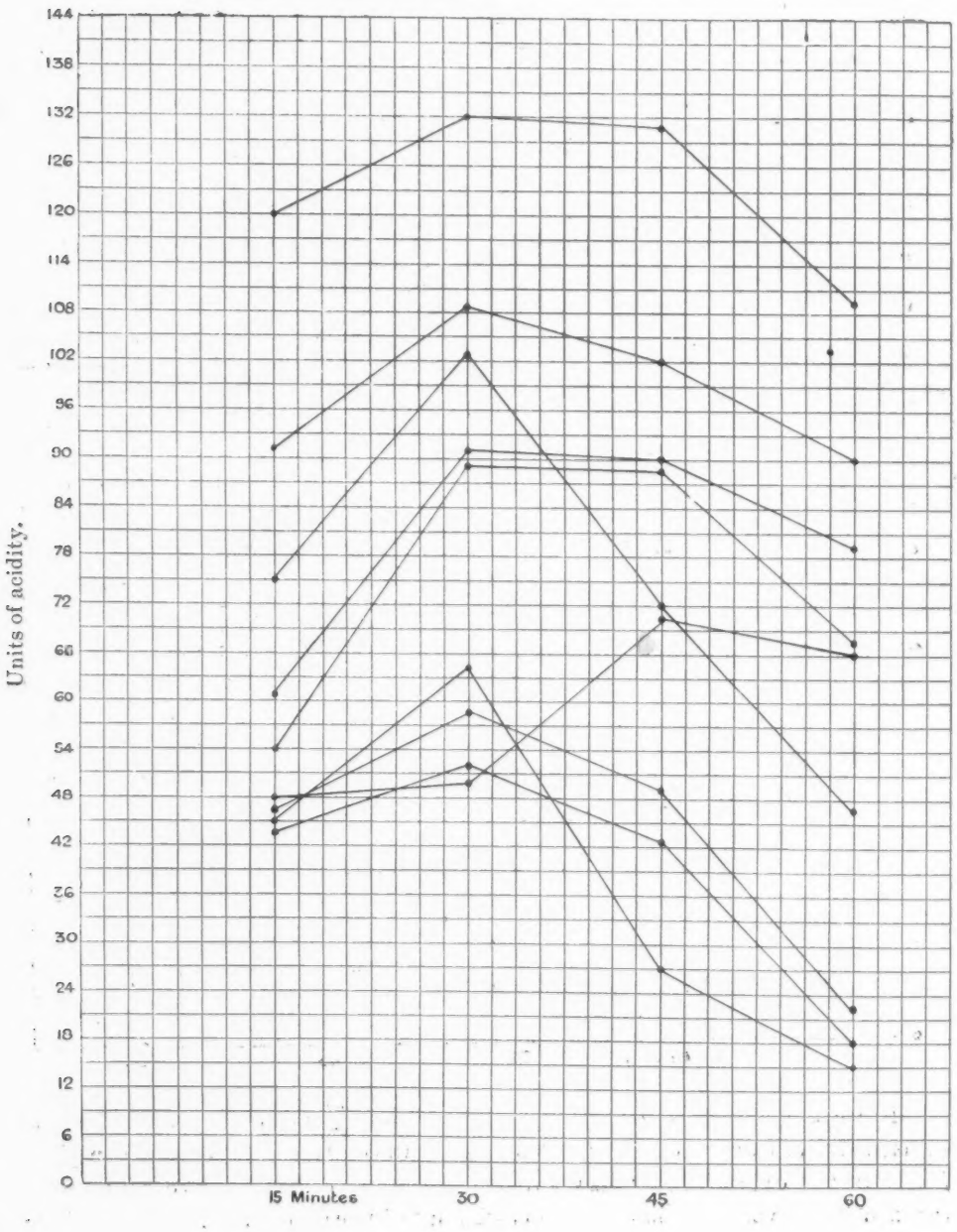


Procedure.—The patient is kept lying down and quiet. Usually the experiment is carried out in the morning immediately following the night's fast. In the case of infants an interval is allowed of four or five hours after the last feed, which should be glucose water instead of milk. Under these

conditions the gastric secretion obtained is devoid of food substances. The tube as previously described is inserted, and the patient allowed to settle down for about a quarter of an hour. Any gastric contents are removed. Histamine is then injected subcutaneously in the dose adequate for the age and size of the patient.

CHART C.

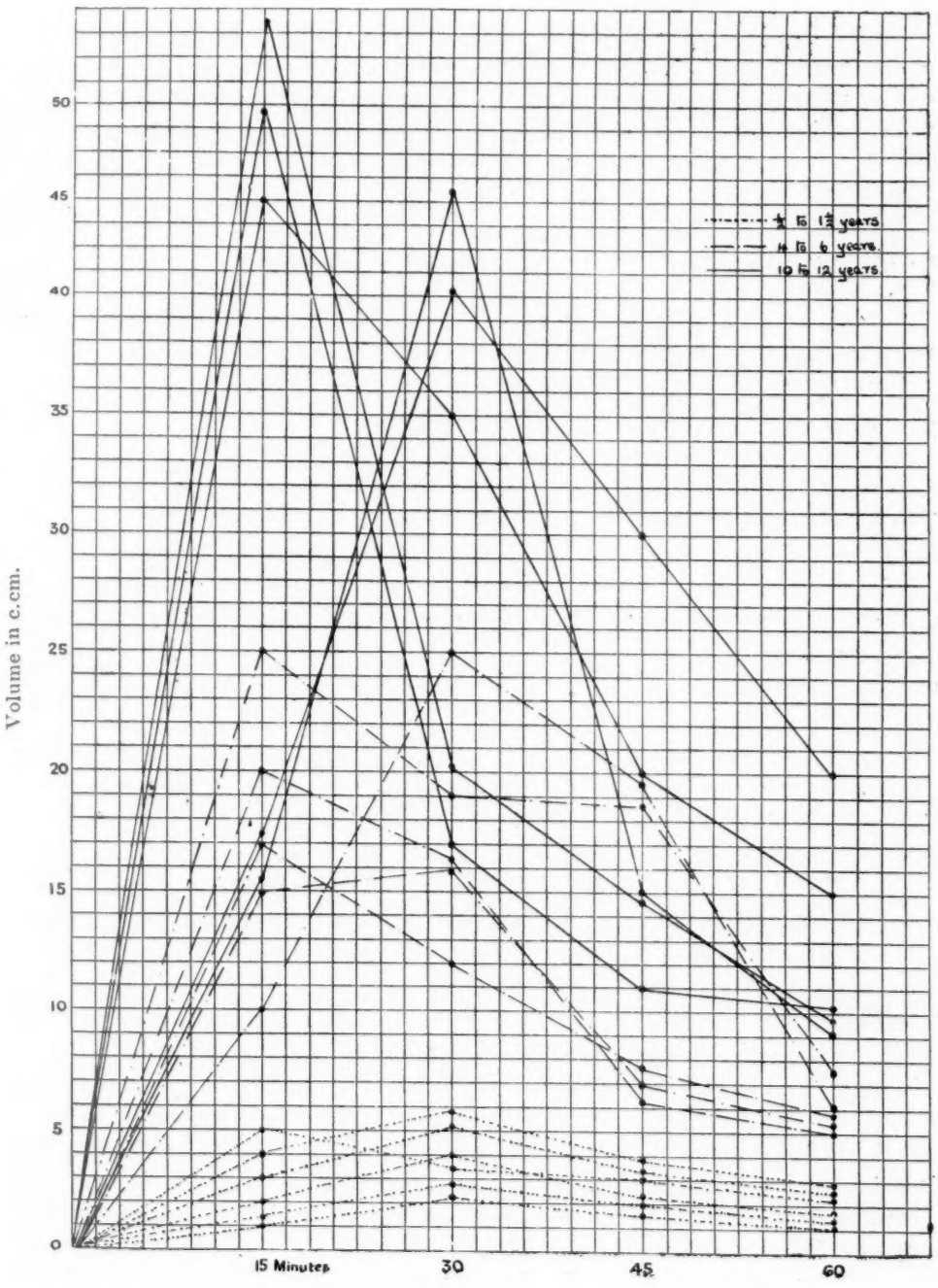
CHILDREN AGED 10 TO 12 YEARS. GASTRIC ACIDITY.



Every fifteen minutes for one hour following injection the stomach contents are removed by suction with a syringe attached to the tube in situ. No disturbance whatever of the patient occurs and gentle movement of the patient on to the left and right sides alternately enables the metal tip to move

CHART D.

VOLUME OF GASTRIC JUICE IN THE THREE AGE GROUPS.



within the stomach and so allows complete removal of all the secretion during the experiment. Since the gastric juice obtained is watery no difficulty is found in its removal. The fifteen-minute specimens are kept separately; each is examined for acidity and its volume also recorded. The pure specimens may be used for the determination of enzymes, base, and nitrogen, if desired. From the data obtained the chloride-secreting power of the stomach and the volumetric response to a fixed adequate stimulus is determined.

Results.—The secretion of acid in the gastric juice of normal healthy children of three distinct age groups is shown in Charts A, B and C. Examination of a large series of cases in each group has been made, but only a number of these are included in the charts. It will be seen that each chart is similar in that in most cases the acidity reaches a maximum within thirty minutes after the histamine is given, and falls thereafter. A few exceptions to this occur, but the fall is always seen within an hour. In each age group there is a considerable range of maximal acidity, although the type of curve is similar in each case.

The volume of gastric juice secreted following the fixed adequate dose of histamine is shown in Chart D. As in the above series, three age groups were examined. Certain outstanding points are quite obvious. During the first year or so of life the total volume does not exceed 15 c.cm. in any case. A considerable rise in volume is obtained in the group aged from 4 to 6 years. Even a greater increase is shown in the eldest group aged from 10 to 12 years. In all the groups the larger volume is obtained during the first half-hour after histamine injection.

In carrying out a number of experiments it was immediately recognized that the physical characters of the pure gastric secretion obtained from the infant are distinctive. The juice is of viscid consistence, somewhat opalescent and odourless. The secretion removed from the older child is watery and clear with a characteristic odour.

DISCUSSION.

Certain comparisons can be made between the acid secretion in children obtained by this method and that obtained by Ryle[5] using the fractional test meal. Although the test meal introduces factors of dilution, buffering and so on, Ryle's series of 100 normal young adults shows the same outstanding fact, namely, the variability of the maximal acid concentration in a group of healthy individuals of similar age. This variation is apparent in all age groups. The results obtained here in children are confirmatory of those obtained by Ryle in adults. It would therefore appear that high or low gastric acidity is a quality inherent in the individual even in very early life. As in Ryle's series, it is probable that a large percentage of cases falls into a medium range and the extreme ranges are in the minority.

What is the significance of the very small volume obtained in the infant and the relatively greater volume in the older child? In several instances, where a tube has been inserted in the duodenum to check the absence of loss of gastric juice from the stomach, 120 c.cm. have been obtained, and in two cases 140 c.cm. were removed in the course of an hour from children aged

about 10 years. It would appear that the larger volume obtained cannot be due simply to the naturally larger stomach in the older child. A more plausible hypothesis would be that the volumetric secretion is a reflection of the nature of the diet at the different ages. It is suggested that the small volume of viscid fluid in the infant is a concentrated medium to allow of adequate dilution by the relatively large fluid volume of the milk diet ; while on the other hand, in the child advanced beyond the milk feeding age, the copious secretion is possibly a provision for the more solid foodstuffs and the adequate formation of the chyme. The small total quantity of acid present in the infant's secretion is rapidly diluted and buffered by the milk foods. This explains the higher acidity in the chyme of the child than in the infant. It is unusual for the reaction of the chyme in an infant to be more acid than pH 4·0 or 3·5. Some unpublished recent experimental work by Esslemont[6] shows that even where the acidity in the infant's stomach is higher than this, the duration of a range of acidity compatible with peptic digestion is only maintained for a short period of time, rarely more than fifteen minutes or half-an-hour. It would therefore appear that the gastric secretion in an infant, being so small in volume and so heavily diluted after feeds, cannot allow of any significant degree of protein digestion in the infant's stomach. Further recent experimental work of the writer has shown that this is compensated for by a high tryptic power of the pancreatic secretion in the infant.

Conclusions.

(1) An easy and accurate method of obtaining the gastric contents in the child is described.

(2) A 7% alcohol-water mixture is an adequate stimulus of gastric secretion in the child and may be used as a test for achlorhydria.

(3) The only adequate test for achylia gastrica is the absence of acid gastric secretion after the subcutaneous injection of histamine.

(4) The maximum acidity of the pure gastric juice varies considerably in any one age group in infants and children. This is similar to Ryle's results in adults using the fractional test meal.

(5) The volume of gastric secretion in infants during the milk feeding period is very small ; after this period of life the quantity is very greatly increased.

(6) It is suggested that the gastric secretion in the infant does not play so important a part in normal digestion as it does in the older child.

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TWO CASES OF VARICELLA ENCEPHALITIS

BY

STANLEY GRAHAM, M.D.

(From the Department of Medical Pædiatrics, Glasgow University, and the Royal Hospital for Sick Children, Glasgow.)

Non-suppurative lesions of the nervous system in association with the common infectious fevers have been recognized as a rare occurrence for many years. Although described clinically on several previous occasions, Barlow and Penrose[1] were the first to demonstrate pathologically the existence of such a lesion. They described the picture of a disseminated myelitis in a fatal case of measles which they had had under their care. Since then small groups of cases have been reported from time to time, not only in measles but in many other infections. Valentin[2] in 1905, collected from the literature 79 cases of paralysis occurring in whooping-cough. In addition, encephalitis or encephalo-myelitis has been described as a complication of smallpox, chickenpox, scarlet fever, diphtheria, mumps, influenza, typhoid fever, typhus fever, syphilis and tuberculosis. Measles and whooping-cough and, more recently, vaccination have, however, accounted for the majority of the cases. That the condition may occur at any stage of the disease, or even as a sequela after convalescence, appears to have been established.

Because varicella is one of the commoner of the infectious diseases and in the majority of instances runs a benign course, it was considered worth while to report the following cases which occurred about the same time and in which the most prominent symptom was ataxia, although signs of pyramidal tract involvement were also present.

Case reports.

Case 1.—I.H., a boy, aged 3 years, was admitted to hospital on January 5th, 1928, because of inability to walk of three days' duration. The previous history was irrelevant. The parents stated that until three days before admission he had been in good health and that they were not aware of any skin eruption.

On examination at this time he was found to be a bright well-built boy who did not appear ill. Crusts and scars of healing varicella in approximately the second week of the disease were present. Contacts of this boy subsequently developed varicella. The most pronounced feature was the presence of an extreme degree of ataxia. There was no paralysis, and no sensory disturbances were detected. The deep reflexes were active, the plantar response was flexor, but poorly sustained ankle-clonus was obtained on the right side. The abdominal reflexes could not be elicited. The pupils reacted to light and there was no nystagmus.

Lumbar puncture revealed a clear fluid under slightly increased pressure. The cell count was 6 and the globulin test (Pandy) was positive. The Wassermann reaction of the blood was negative.

On February 20th, although the ataxia was still marked, the boy seemed in every other way perfectly well and was dismissed home. He was seen again on March 22nd, and was perfectly well. His mother stated that all unsteadiness disappeared within a fortnight after dismissal from the ward.

Case 2.—R.P., a boy, aged 6 years, developed varicella on May 15th, 1928, a mild attack with few constitutional symptoms. On June 3rd (i.e., nineteen days after the onset of the varicella) he went to bed quite well, but next morning he vomited, and on attempting to get out of bed found that he could not stand without support and 'swayed about like a drunk man.' This state of affairs persisted and on June 15th, he was admitted to hospital.

On examination, he was a well-nourished boy who did not look ill. He walked with a markedly ataxic gait and tended to fall to the right. Both legs were hypertonic, with weakness of the right. The knee-jerks were exaggerated, the plantar response was extensor on the right side and flexor on the left, and ankle clonus was present on both sides. No sensory disturbances were detected. There was no ocular paralysis or nystagmus. There was no swelling of the optic discs although some slight fullness of the veins was noted.

Lumbar puncture showed a clear colourless fluid under slightly increased pressure. The cell count was 9 (all lymphocytes), and there was no increase in globulin. The Wassermann reaction of the blood was negative.

On July 11th he was able to walk quite well, although slowly and with care. The knee-jerks were still very active, but the ankle clonus had disappeared and the plantar responses were flexor. When seen again some weeks later nothing abnormal could be detected.

Discussion.

Two views are expressed regarding the ætiology of these nervous manifestations occurring in the infectious fevers. One is that the encephalitis and the specific fever are coincident infections, the nervous lesion being predisposed to in some way by the presence of the other disease. The other theory is that the encephalitis is the result of the action of a virus or toxin elaborated by the organism producing the infectious disease. This suggestion of a neurotropic virus is not new but has not yet received ready acceptance. The apparently increasing incidence of encephalitis following vaccination has again, however, brought the subject under discussion. Strumpell[3] in 1884 suggested that just as polio-myelitis affects the anterior horn cells of the cord, so may the brain be affected by the disease which he called polio-encephalitis. In the next year, however, Marie[4] suggested that the virus producing the infantile paralysis was responsible in only a certain percentage of cases of polio-encephalitis, and he stressed the association of encephalitis and the specific fevers, and indicated that the toxin of the specific fever might be the causal agent. This view has since been expressed by numerous writers. Leichtenstern[5] in particular believed this to be the case, and both he and Nauwerk[6] reported their observations on the occurrence of the disease during the pandemic of influenza at the end of last century.

Greenfield[7], when recently discussing the question in connection with measles, suggested the possibility of the condition being caused by some unknown virus which spreads in epidemic waves and only produces disease when brought into activity by the exanthem. He believes that the groups of reported cases appearing at distinct intervals support this idea. It may be a significant fact that the two cases described above were observed within six months. Considering the prevalence of varicella, and its usual freedom from complications, this lends some slight support to Greenfield's suggestion.

Histologically it would appear that the lesions in encephalitis fall into two main groups. The one group, which includes polio-encephalitis and encephalitis lethargica, is characterised by the presence of well-marked perivascular 'cuffing.' In the other group embracing all the encephalitic manifestations of the virus diseases including the post-vaccinal type, the cuffing although present is not so marked, the characteristic picture being produced by the presence of definite zones of perivascular demyelination. McIntosh[8], however, goes so far as to say that investigations of these virus infections show that a histological picture of a certain type can be assigned to each. He also mentions that in the post-vaccinal type for example, the only toxin which has been discovered is the vaccinal one and that certain strains will produce in rabbits an encephalitis of the post-vaccinal variety. These arguments are strongly in favour of the theory that the toxin of the specific fever is the ætiological agent.

One of the main difficulties in arriving at a complete acceptance of either theory is the paucity of post-mortem material. The morbid anatomy of varicella encephalitis is, for example, unknown, no fatal case so far having been reported.

From a perusal of the literature, it would appear that great variations in the course and prognosis may occur. Many cases obviously recover completely. Apart from the cases ending fatally in the acute stage, such conditions as hemiplegia, paraplegia, athetosis and mental defect have been described as persisting. Deafness has also been mentioned but in such cases, particularly those complicating measles, it is necessary to consider the possibility of the deafness being due to a labyrinthitis.

In varicella encephalitis the prognosis appears to be good. All the recorded cases probably number less than twenty. These have been collected in papers published by Miller and Davidson[9], and more recently by Ford and Wilson[10]. As previously mentioned, no fatal case has as yet been reported.

Conclusions.

1. Two cases of varicella encephalitis are reported.
2. Evidence so far obtained goes to show that in the encephalitis associated with the virus diseases the ætiological agent is probably the virus of the specific infection.
3. In varicella encephalitis the prognosis is favourable.

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A CLINICAL AND BIOCHEMICAL STUDY OF THE SUBNORMAL CHILD

BY

A. ARNOLD OSMAN, M.R.C.P.,

and

HAROLD G. CLOSE, M.B., B.S.

(From the Medical Investigation Dept., Queen Mary's Hospital, E.)

The subjects discussed in this paper are the results of an investigation primarily undertaken with a view to determining as far as possible some of the biochemical factors underlying, and predisposing to, the various forms of nephritis in children and adults. In the course of our investigations, it soon became evident that the problem resolved itself into a study of subnormal health or 'debility,' as predisposing to disease in general; or, in other words, into a a study of the nature of the soil upon which disease flourishes. In this paper it is proposed to present the results of the investigation in so far as they relate particularly to children. The results of parallel observations on adults are being published elsewhere[1]. It is hoped too, that some of the results obtained may form a standard upon which to base further observations on specific diseases as they occur in children, for not a few biochemical data,published as representing the variations peculiar to specific diseases, are in fact already present in that state of subnormal health. This so frequently precedes frank disease that it in part explains the similarity in the findings in diseases which are, in their pathogenesis and clinical nature, widely different.

Selection of cases.—The children investigated were all of the elementary day-school social class, who attended the medical out-patient department of one of us (A.A.O.), at Queen Mary's Hospital, for such minor symptoms as headache, lassitude, constipation, and bilious attacks. The actual symptoms for which advice was sought are given in Table 1. The children were all between the ages of six to twelve years inclusive. Those who had recently suffered from some acute illness, such as tonsillitis or measles, were not included.

The normal control children were selected from those patients who attended the casualty department for such conditions as ringworm, or uninfected cut fingers, and who appeared in every other way to be in perfectly normal health. A certain number of normals was also recruited from among the families of patients.

In this way it was found possible to divide the cases roughly into two groups clinically, the normals, and the subnormals or 'debilitated.' A certain amount of overlapping between the groups inevitably occurred, as it was desired particularly to investigate that borderland state between health and

disease, where the one passes almost imperceptibly into the other, but as will be seen later, subsequent biochemical observations to a large extent confirmed and justified this method of selection.

With few exceptions, a sample of blood for examination was taken between two and three hours after the last meal, the nature and constitution of which was ascertained. Measures were also adopted to ensure that each child was resting quietly for at least an hour before the blood was taken, and of course, in no case was the child aware that this operation was to follow. After considerable experience in obtaining blood from children, it is considered that the technique employed is so free from causing pain, and even but momentary apprehension, that changes in blood chemistry from this cause can be excluded. In a few instances where the element of fright was encountered, the case was excluded from the series.

Methods.—The following methods were used in this investigation :—

(1) Plasma bicarbonate, by the method of Van Slyke, Stillman and Cullen[2]. Titration was carried to a constant end-point of pH = 7·4. Phenol red was used as indicator. Results are expressed as molar concentration of bicarbonate.

(2) Plasma chlorides by the method of Claudius[3], results being expressed as grammes of NaCl per 100 c.cm.

(3) Ketone bodies in the blood by the method of Van Slyke and Fitz[4].

(4) Blood sugar (venous blood), by the method of Folin and Wu, results being expressed as milligrammes per 100 c.cm.

Procedure.—The following procedure was adopted, by means of which it is hoped that any personal bias in correlating the biochemical and clinical results, has been reduced to a minimum.

Each child was submitted to a thorough clinical examination by one of us (A.A.O.). This included inspection of the tonsils, etc., and a qualitative examination of the urine for albumen, sugar, and acetone bodies (Rothera and ferric chloride reactions). These results, with the history of the case, and notes on signs or symptoms, were entered up, and a diagnosis of either 'normal,' or 'subnormal' was made. The case was numbered and the child was given a card bearing this number only.

The collaborator (H.G.C.), who had not previously seen the child, then took, without undue compression, a sample of blood under paraffin from a vein in the arm into an oxalated centrifuge tube, which was correspondingly numbered. Estimations were performed within twelve hours of taking the blood, it having been found previously that none of the quantities estimated showed appreciable variations within this period. The blood sugar, however, was estimated immediately after taking the sample of blood.

Comparison of normal and subnormal children.

The main clinical features and chief symptoms of all the children investigated in this series are shown in Table 1. A few of the headings in this table require explanation. Cyclical vomiting ('C.V.') was diagnosed only when there was a clear history of recurrent 'bilious attacks,' preceded by listlessness, and accompanied by headache, vomiting and prostration, lasting 48 hours or more, and necessitating confinement to bed for this period. Debility ('D') was diagnosed in those children who were in subnormal health, but who did not give a clear history of attacks of cyclical vomiting. Children who were in a permanent state of debility, and who also suffered from cyclical vomiting, have been classed under the heading of C.V. only.

The nomenclature adopted in assessing the condition of the tonsils was as follows:—Cases in whom a complete tonsillectomy had been performed at least a year previously, were labelled (a); those in whom there was no history at any time of throat or ear trouble, and whose tonsils appeared to be normal, were labelled (b); and those who gave a history of repeated throat troubles, i.e., tonsillitis, 'sore throats,' cervical adenitis, with or without chronic otorrhœa, and whose tonsils either appeared unduly large, or definitely septic, were labelled (c). No doubt this classification is open to criticism, but it was the best that could be devised, and the number of cases is sufficiently large to make it at least of some value.

An attempt was also made to assess roughly the temperament of each child, that is, whether or not there was an undue tendency to nervousness, excitability, or the reverse. In doing so it was only possible to form an opinion from the behaviour of the child under examination, and from the parent's statements regarding this—neither of them very reliable guides.

Table 1 shows that 108 children were investigated, 38 normals, and 70 subnormals.

Table 2 compares the blood sugar, plasma bicarbonate, and chloride, in groups of normal and subnormal children.

From a consideration of these figures (Table 2), three important facts emerge. First, the plasma bicarbonate is considerably lower in the subnormal than in the normal children. In fact, a definite 'acidosis' is the rule in debilitated children, if the term 'acidosis' is defined as simply a decrease in the plasma bicarbonate, however produced. Such a definition does not necessarily imply any actual change in reaction, or pH, in the direction of increased acidity, in the strict chemical sense of the word. Secondly, this acidosis of debility, is associated with a slight, but definite, increase of the plasma chloride, compared with that of the normals. This point is of interest in view of the experiments of Haldane[5] and his co-workers, who showed that after ammonium chloride ingestion, the plasma bicarbonate was decreased in exact proportion as the chloride content increased; and also because of the possibility of an accompanying increase in the water content of the body, a subject which has been more fully discussed elsewhere.[1] Thirdly, it is interesting to note that the blood sugar is practically the same in both groups. This is contrary to recent teaching, which asserts that the subnormal child shows a tendency to continued hypoglycæmia—an hypothesis which seems based on insufficient evidence.

One of us (A.A.O.) when working on the subject of cyclical vomiting in children some years ago, showed that this common disorder could generally be prevented or cured by increasing the allowance of sugar in the diet, and it was assumed that there was therefore a shortage of sugar in the blood. The term 'glycopenia,' which unfortunately is still used by some, was coined to describe these children. It is clear, however, from the evidence already presented, that this term is inaccurate, and should no longer be used in this connection. As has been pointed out before[6], the essential feature of these cases appears to be a shortage of the glycogen stored in the liver, and perhaps

TABLE 1. COMPARISON OF NORMAL AND SUBNORMAL CHILDREN.

Case	Sex	Age	Height		Weight		Diagnosis	Chief symptom	Constipation	Headache	Tonsil history	Disposition
			ft.	in.	st.	lb.						
1	M	10					N			+	b	Bright
2	F	9					SN	C.V.		+		Dull
3	F	13					SN	Languor		+		Dull
4	M	8					SN	C.V.				Normal
5	F	6½					SN	C.V.		+	c	Normal
6	F	9					N	C.V.				
7	M	8					SN	C.V.				
8	F	7½					SN	Headaches		+		
9	F	7					SN	C.V.				
10	M	5½					SN	C.V.				Dull
11	F	6					SN	C.V.				
12	M	6			2	10½	SN	C.V.				
13	F	6			2	8½	N	Languor		—		Dull
14	M	8					SN	Languor		+		Dull
15	M	8	4	1½	4	0½	N					Normal
16	M	7½					SN	Languor				Dull
17	M	12					N	C.V.				
18	M	12					SN	C.V.		c	c	Normal
19	M	8					SN	Languor				Excitable
20	F	12					SN	C.V.			c	Excitable
22	M	13					SN	C.V.		+		
23	M	6					SN	C.V.				
24	F	12	4	6¾	4	8½	N	C.V.		+	b	
25	M	12			7	3	SN		—	+	b	Dreamy
26	M	12			5	1	N	Recurrent abdominal pain.	—	+	b	Dull
27	M	10			5	1½	N	Recurrent abdominal pain.			b	
28	F	8					SN	Joint & limb pains		+	b	
29	M	5			2	6½	SN	C.V.		+	c	
30	F	13	4	4½	8	4½	SN	C.V.		++	a	
31	F	7	3	11	3	3½	N	Threadworms and C.V.			c	
32	F	10			4	0	N	Giddy attacks	+	+	b	Dull
33	F	8½			4	12	SN	C.V.			a	Excitable
34	F	6			4	13	SN	Wasting and C.V.			b	
35	M	11	4	5¼	4	8	SN	Abdominal pain and C.V.		+	a	
36	M	8					N				b	Normal
37	M	5½					SN	C.V.; drowsiness	—		c	Dull but excitable
38	F	8			3	1	SN	C.V. & excitability		+	b	Excitable
39	F	8			3	6	SN	Debility		—	a	Excitable
40	M	7	3	10	2	13½	N	Cough		+	c	Excitable

Habit	Complexion	Remarks	Plasma $NaHCo_3$	Plasma NaCl	Blood sugar	Rothera urine	Urinary chloride
Fat & flabby	Pasty	Suffers from C.V.	·0306			—	
Normal	Pasty		·0270			sl. +	
Normal	Good		·0270				
					·107		
Normal			·0280		·09	—	
Normal	Good		·0305		·108	—	
			·0277		·079	—	
			·0278		·093	—	
			·0267		·085	—	
Frail	Pale		·0275		·146	—	
			·0223		·078	—	
			·0288			+	
Normal	Pale	Growing pains	·0308		·089	—	
Normal	Pale		·0302		·125		
			·0292		·139	—	
	Pale		·0263		·101	—	
		Suffers from angio-neurotic œdema.	·0302			—	
Normal	Pale		·0290		·114	++	
Thin	Pale	Attended for ? chorea (not confirmed).	·0269		·130	—	
Thin	Pale	Appendicectomy 2 years ago	·0271		·116	—	
					·105		
					·075		
Normal	Good	Sick in trains, etc.	·0290		·083	—	
Fat & flabby	Good	Septic sores on body	·0262		·103	—	
Fat	Good		·0292	·614	·081	—	·985
Normal	Good		·0299		·077	—	
Thin	Pasty		·0287		·099	—	
Thin	Good		·0273		·090	+	
	Pale	Tonsillectomy 8 years before	·0303		·102	—	
			·0135		·016	—	
	Pale		·0290		·081	—	
	Pale	Tonsillectomy 1 year ago	·0281		·092	—	
			·0279			—	
	Pale	Appendicitis simulated. Tonsillectomy 7 years ago.	·0285		·109	—	
Normal	Good		·0309		·102	—	
Normal	Pale		·0275		·093	+++	
Thin	Pale	Pains in limbs in damp weather	·0316		·079	—	
Thin	Pale	C.V. Tonsillectomy 3 years ago.	·0302			—	
Thin	Fair		·0280			—	

TABLE I—*continued.*

Case	Sex	Age	Height		Weight		Diagnosis	Chief symptom	Constipation	Headache	Tonsil history	Disposition
			ft.	in.	st.	lb.						
41	F	7			2	9	SN	Frequent colds		+	c	Normal
42	F	5½			2	13	SN	T's & A's—C.V.	—	+		Excitable but drowsy
43	M	12	4	9½	5	7	N			+		
44	F	9			3	1½	SN	Anorexia			c	Dull
45	F	9	4	4	3	10½	SN	Debility			b	Dull
46	M	8	3	10	3	2½	SN	Asthma	+		a	Dull but excitable.
47	M	8	4	2	4	1	SN	Pains in chest and	+	+	a	Dull
48	F	8	3	10	3	0½	N	Tram-sickness		+	c	Dull
49	M	13	4	4	4	10	SN	Headaches			b	Dull
50	F	10	4	5	4	1	SN	Abdominal pains	+	+	b	Excitable
51	F	5			2	7	SN	Frequent headaches		+	a	Normal
52	F	10			4	3	N	Frequent headaches			a	
53	F	13	4	10	6	0	N	Cough		+	c	Normal
54	M	8	4	1½	3	7½	N	? heart disease	—	—	b	Normal
55	M	5½	3	7¾	3	0½	SN	Languor, anæmia	+	+	c	Normal
56	M	7½	4	0¼	3	10½	SN	Boils and cough	—	+	b	Excitable
57	M	8½	4	1	3	7	SN	Lassitude	+	+	a	Excitable
58	M	8	3	11½	3	4½	SN	C.V.	+	+	b	Excitable
59	M	6¾			3	8	SN	Lassitude	+	+	c	Dull
60	M	9½			4	9½	N		—		a	Normal
61	M	8			2	13½	SN	Asthma	—	—	b	Excitable
62	F	9			4	7	SN	Abdominal pains	+	+	a	Excitable
63	M	10	4	5½	4	13	N	Boils	—	+	a	Excitable
64	F	10	4	4	3	13½	SN	Growing pains	—	+	a	Excitable
65	F	9	4	1¼	3	3	SN	Boils	+	+	c	Excitable
66	M	5½	3	8	3	0	SN	Anorexia	—	—	b	Normal
67	M	11	4	10	6	2	SN	C.V. and boils	—	—	b	Normal
68	M	8	4	3	4	6	SN	C.V.	+	+	b	Excitable
69	F	9			4	9	SN	Anæmia	—	—	a	Phlegmatic
70	M	11	4	3½	4	7	N	C.V.	—	+	b	Normal
71	M	13	5	3½			SN	Listlessness	+	+	a	Normal

Habit	Complexion	Remarks	Plasma $NaHCo_3$	Plasma NaCl	Blood sugar	Rothera urine	Urinary chloride
Thin	Pale		·0260			—	
Normal	Pale		·0269			+—	
Thin	Pale	Nervous—afraid of dark	·0321			+—	
	Pale	C.V.	·0248			+—	
	Pale		·0290			—	
Thin	Pale	Tonsillectomy 3½ years before, also previous C.V.	·0280	·594		—	
Thin	Pale		·0275			—	
Normal	Good	Nervous	·0314			—	
	Pale	Pains in feet. Old rheumatic heart.	·0282			+	
Thin	Good	Anorexia	·0287			—	
Normal	Good	Pains in all joints 2 years ago. Tonsillectomy 2 years ago.	·0268	·632		—	
Thin	Good	Nervous. Tonsils out a year ago.	·0312	·608		—	
Normal	Good	Sick in train	·0314	·632		—	·655
Normal	Good	Ac. rheumatism 2 years ago	·0287	·594		—	·702
Normal	Good	Train sickness. ? occasional C.V.	·0289	·638		—	·288
Normal	Pale	Train sickness and C.V.	·0277	·708		—	1·12
Thin	Pale	Train sickness. Tonsillectomy 1 year ago.	·0275	·623		—	1·08
Thin	Good		·0316	·611		—	·614
Normal	Pale		·0283	·608		+	·80
Normal	Good	Tonsillectomy 4 years ago	·0302	·597		—	·535
Normal	Good		·0277	·605		—	·807
Normal	Pale	Is train sick. Tonsillectomy 2 years previously, also suffers from C.V.	·0294	·620		—	·625
Normal	Good	Tonsillectomy 5 years previously, also has cyclical vomiting.	·0296	·587		—	·526
Normal	Good	Suffers from C.V. Tonsillectomy 2 years before.	·0268	·605		—	·658
Thin	Pale	Train sick	·0264	·617		—	·646
Normal	Very pale		·0266	·611		—	·714
Normal	Good		·0271	·603		—	·453
Normal	Pale	Acute rheumatism twice last year. Growing pains and C.V. Heart normal	·0284	·620		—	1·14
Normal	Pale	Tonsillectomy 1 year previously.	·0296	·623		—	·49
Normal	Good		·0294	·638		—	·439
Normal	Pale	Formerly C.V. Tonsillectomy 6 years previously	·0283	·594		—	·530

TABLE I—*continued.*

Case	Sex	Age	Height		Weight		Diagnosis	Chief symptom	Constipation	Headache	Tonsil history	Disposition
			ft.	in.	st.	lb.						
72	F	8	3	9	3¼	1	N	Headaches	—	+	c	Normal
73	F	10	3	6	4	3¼	N	Thinness	—	—	a	Excitable
74	F	13	4	2¼	4	1	SN	Pains in legs	+	—	b	Normal
75	M	9	4	2¾	4	6	N	C.V.	+	+	c	Normal
76	F	9			4	12	N	Nerves	+	—	c	Excitable & phlegmatic
77	M	12	4	10	5	1	SN	Headaches	—	+	b	Phlegmatic
78	F	7½	3	9	3	3	SN	Headaches	—	+	c	Excitable
79	M	7	3	9½	2	13½	SN	Catarrhs	—	+	a	Normal
80	M	7	4	1½	3	9	N	Nerves	—	+	b	Very excitable
81	M	7	3	8	2¾	13½	N	C.V.	—	+	c	Normal
82	M	7	3	5½	2	12½	N	C.V. & headaches	—	+	b	Nervous
83	F	9½	4	1	4	2	SN	C.V. and pains in legs and arms.	+	+	a	Normal
84	F	8	4	1¾	3	7	SN	Rheumatics	—	+	c	Excitable
85	F	8	3	10¾	2	13	SN	Abdomen pain and headaches	+	+	c	Excitable
86	M	8	4	3½	4	0	SN	Cough and headaches.	—	+	b	Normal
87	F	8	4	2¾	4	2½	N	Headaches	—	+	b	Excitable
88	F	7	4	4½	4	8½	N	C.V. and ? rheumatic pains	—	+	b	Normal
89	M	8½	3	11	3	4	SN	C.V.	—	+	a	Normal
90	M	9	4	3½	3	13½	SN	Constipation	+	+	c	Phlegmatic
91	F	6			2	12¼	N	Night terrors	+	+	c	Excitable
92	F	8	4	1¾	4	0	N	T.T.	—	—	c	Excitable
93	F	11	4	6	4	12¼	N	Pains in back	—	+	c	Normal
94	F	8	3	11½	3	6½	N	Headaches and ? rheumatic pains.	—	+	b	Excitable
95	M	7½			3	6¼	SN	Wasting, 'backwardness.'	—	+	c	Normal
96	M	7½					SN	Pains in knees	—	—	b	Normal
97	M	6½					N	C.V.	—	+	b	Normal
98	M	9	4	1¾	3	8	SN	Cough	+	+	b	Normal
99	F	7	3	10½	3	0	SN	Cough	+	+	c	Excitable
100	F	6½	3	5½	2	6	SN	Fidgets	+	+	c	Excitable
101	M	9	4	5¾	4	2	N	Headaches	—	+	c	Normal
102	F	7	3	10	3	6	N	Urticaria papulasa	+	+	b	Excitable

Habit	Complexion	Remarks	Plasma $NaHCO_3$	Plasma NaCl	Blood sugar	Rothera urine	Urinary chloride
Normal	Good		·0293	·588		—	
Thin	Pale	Had growing pains. Tonsillectomy 5 years ago	·0295	·590		—	
Normal	Good	? subacute rheumatism; slight systolic bruit apex.	·0270	·608		—	1·14
Normal	Pale	Train sick formerly	·0303	·605		—	1·16
Normal	Good		·0295	·600		—	·32
Thin	Pale	Train sick	·0303	·603		sl. +	1·12
Thin	Pale	Has cyclical vomiting	·0292	·629		sl. +	1·14
Thin	Pale	Tonsillectomy 3½ years previously.	·0291	·588		++	1·08
Normal	Good		·0311	·604		—	·506
Thin	Good		·0292	·620		—	·111
Normal	Good	Extreme nervousness ? rheumatic pains. Heart normal.	·0263	·611		—	1·18
Normal	Pale	Train sick. Tonsillectomy 1½ years ago. Urticaria papulosa.	·0288	·614		—	
Thin	Good	Heart ? slightly enlarged, no murmurs.	·0270	·600		+—	·687
Normal	Pale	Train sick	·0308	·603		—	·06
Normal	Good	Wets bed	·0254	·614		—	·995
Normal	Good		·0288	·618		—	·664
Normal	Good	Heart normal	·0294	·603		—	1·14
Very thin	Good	Train sick. Tonsillectomy 3½ years ago.	·0228	·598		—	1·21
Normal	Pale		·0264	·607		++	·658
Normal	Good		·0294	·606		—	·589
Normal	Good		·0299	·603		sl. +	·679
Normal	Good	Train sick	·0306	·608		—	1·05
Normal	Good	Heart normal. Had ? rheumatic fever 2 years ago.	·0296	·597		—	·326
Normal	Good		·0284	·611		sl. +	·401
Normal	Good		·0278	·591		—	
Normal	Good		·0300	·605		—	·393
Thin	Pale	Train sick. Systolic bruit at apex.	·0278	·576		—	·439
Thin	Pale	C.V.	·0245	·603		—	·907
Thin	Pale		·0257	·617		—	·890
Thin	Good		·0315	·611		—	·962
Normal	Good		·0299	·594		+	·965

TABLE I—*continued.*

Case	Sex	Age	Height	Weight	Diagnosis	Chief symptom	Constipation	Headache	Tonsil history	Disposition
			ft. in.	st. lb.						
103	F	12	4 6	4 7	SN	Anæmia	+	—	b	Phlegmatic
104	F	[8			SN	Abdominal pains	—	+	a	Phlegmatic
105	M	[6	3 7¼	2 8½	SN	Abdominal pains	+	+	b	Excitable
106	M	10	4 4	4 3½	SN		+	—	c	Excitable
109	M	7	4 0½	3 8½	SN	Headaches		+	c	
110	M	6	3 9¼	3 1½	SN	? fainting attacks	+	—	b	Phlegmatic
119	M	8			SN	Asthma		+	a	

elsewhere, and not a deficiency of sugar in the blood. To summarize the findings in these two groups therefore, it may be said that in subnormal, or debilitated children, there is a definite acidosis, with a tendency to high plasma chlorides, and no evidence of hypoglycæmia.

TABLE 2.

COMPARISON OF NORMAL AND SUBNORMAL GROUPS.

Type	Blood sugar (mgrm. %)			$NaHCO_3$ (molar)			NaCl (grm. %)		
	No. of	Av.	Range	No. of	Av.	Range	No. of	Av.	Range
Normal	11	95	77—139	38	·0299	·0263—·0321	24	·605	·587—·638
Subnormal	25	100·6	78—146	70	·0276	·0228—·0316	40	·610	·576—·659

Acidosis not due to ketosis.—Owing to the frequency with which acetonuria occurs in sick children, it has generally been assumed that the acidosis so commonly found in them, is due to excess of ketone bodies in the blood. In fact, the terms ketosis, acetonæmia, and acidosis, have long been used synonymously by clinicians. Frew[7] has shown that something like 60% of children entering hospital for various conditions have acetonuria on admission, and this high incidence has been confirmed by others. But it must be remembered that all these children are sick, and many of them pyrexial. Acetonuria is almost constantly present too throughout the earlier stages of the acute exanthemata, such as scarlet fever, measles and diphtheria, and this is usually accompanied by some degree of acidosis (decreased plasma bicarbonate). As a rule, however, even in the acute exanthemata, the amount of acetone bodies in the blood is not sufficient to account for the

Habit	Complexion	Remarks	Plasma $NaHCo_3$	Plasma NaCl	Blood sugar	Rothera urine	Urinary chloride
Thin	Very pale	Train sick. Tonsillectomy 3 years previously.	·0278	·588		+	·994
Thin	Very pale	Simulating appendicitis	·0263	·614		sl. +	1·32
Thin	Good		·0280	·638		—	
Thin	Pale	Rheumatic fever 15 months ago. Mitral regurgitation.	·0276	·612		—	·396
			·0278	·588		—	1·26
Flabby	Pale		·0274	·601		—	·58
Normal	Good	Tonsillectomy 3 years previously.	·0272	·600		—	

whole of the decrease in the plasma bicarbonate : in other words, the whole of the acidosis is not due to ketone bodies[8]. Our observations on subnormal children suggest that the acidosis which is so frequently found, is practically never due to an associated ketonæmia. The evidence for this statement is as follows :—

(a) Of the total 108 cases, a positive Rothera reaction was found in the urine at the time of the blood examination in only 17 cases, 1 normal, and 16 subnormals (i.e., 16·5 per cent.). A positive ferric chloride test was present in none of the cases.

(b) Table 3 shows the chemical findings in the cases in whom a positive Rothera reaction was found in the urine. Comparing the figures in Tables 2 and 3 it is seen that in the acetonuric children, the average plasma bicarbonate was even a little higher than the average for the group (i.e., subnormals), namely, 0·0280 M, compared with 0·0276 M. It will also be observed that the blood sugar in the acetonuric and non-acetonuric children is practically the same, namely, 99 mgrm. per cent. in the former, and 100·6 mgrm. per cent. in the latter.

TABLE 3.

CASES SHOWING ROTHERA REACTION IN THE URINE.

Type	Blood sugar (mgrm. %)			$NaHCO_3$ (molar)			NaCl (grm. %)		
	No. of cases	Av.	Range	No. of cases	Av.	Range	No. of cases	Av.	Range
Normal	—	—	—	1	·0299	—	1	·594	—
Subnormal	3	99	90—114	16	·0285	·0248—·0303	9	·608	·588—·632

It is clear then that in the debilitated child the presence of absence of acetonuria furnishes no guide to the degree of acidosis.

(c) In a study of a large number of cases of scarlet fever, diphtheria, measles, typhoid, and streptococcal tonsillitis[8], in which repeated estimations of the plasma bicarbonate were carried out at regular intervals throughout the course of the illness, it was found, as noted above, that estimations of the total acetone bodies in the blood, even in the early and pyrexial stages of the disease during which acetonuria was present, showed that the acidosis was not nearly accounted for by the quantity of these substances present in the blood. Further, it was shown that in 25 per cent. of cases of scarlet fever, a late or delayed acidosis occurred, often as marked in degree as the primary or early acidosis; and that this late form of the condition, which generally occurred between the 15th and the 30th days of the disease, was not accompanied by acetonuria.

(d) In the apyrexial subnormal children with acetonuria in the present series, an attempt was made to estimate the total acetone bodies in the blood of four cases. In none of them was it possible to obtain any evidence of ketonæmia, in fact, no more precipitate was formed than in the 'blank' estimations. On these grounds therefore it is concluded that the acidosis of debility is not due to ketosis.

In this connection, it is of interest to note the difference in response to treatment with sugar, between the apyrexial subnormal child with or without acetonuria, and the child suffering from acute pyrexial disease, such as scarlet fever. In the former, with a normal blood sugar (see above), the giving of extra sugar by mouth usually produces a rapid and striking improvement in the general health[6]; whereas in the latter, where the blood sugar tends if anything to be above normal, not only does the giving of sugar usually fail to produce any improvement in the condition of the child, but it does not hasten the disappearance of acetone bodies from the urine. This suggests that during acute pyrexial disorders, there is inability to utilize the sugar: whereas in debility, although the blood sugar is normal (it is presumably of the first importance to maintain a normal concentration of sugar in the blood), the glycogen reserves of the body are on the verge of exhaustion.

Before leaving the subject of the biochemical variations in the two groups of children investigated, it may be of interest to cite a few of the individual cases, as illustrating some of the points already raised. It must be emphasized that although a decreased plasma bicarbonate is the rule in debilitated children, the degree of acidosis is not necessarily a measure of the unfitness in any given case, though there is a rough parallelism between the two factors. Case 82 looked in every way in the most perfect health, yet he had a plasma bicarbonate of only 0·0263 M. Case 18, on the other hand, looked extremely unwell, had a strong positive Rothera in the urine, and a plasma bicarbonate of 0·0290 M. The relation between the blood sugar and the degree of fitness is of special interest, in view of the current teaching to which reference has already been made. The children who appeared to be most unfit, and were pallid, exhausted, and lacking in energy and in interest in their surroundings,

were not those with the lowest blood sugars, but, on the contrary, it was quite often found that their blood sugar was relatively high, even above normal. This was particularly noticeable in Cases 10 to 16, 18, 19, 20, 25, and others. Further, repeated estimations of the blood sugar at fortnightly intervals on some of these cases, failed to show any more 'instability' in the blood-sugar level in the one group than in the other. No useful purpose would be served by multiplying instances of these discrepancies, they all go to show that isolated observations of this kind are of little value, and that individual differences are so marked, that it is only in the study and comparison of groups of similar cases, that much can be learned by our present rather crude methods.

Relation of tonsillar disease to debility in children.

Disease of the tonsils and of the upper respiratory tract in general, has so often been considered as one of the chief causes of debility in children, that the removal of tonsils and adenoids is now regarded as almost a necessary preliminary, if not the only treatment required in these cases. It was considered desirable, therefore, to assess as far as possible the relation of tonsillar disease to the clinical and biochemical findings in the present series of cases.

In 94 children, 35 normals and 59 subnormals, the state of the tonsils, and the history of tonsillar disease, including sore throats, cervical adenitis, and chronic otorrhœa, were carefully noted. Table 4 shows the state of the tonsils as found in the healthy and debilitated children. The notation used is that described previously.

TABLE 4.

CONDITION OF TONSILS IN CASES EXAMINED.

	A.	B.	C.
Normal	5	16	14
Subnormal	17	21	20

A study of Table 4 shows that :—

(1) of 30 normal children, approximately 50 per cent. had tonsillar disease, and 50 per cent. did not.

(2) of 42 subnormal children, approximately 50 per cent. had tonsillar disease, and 50 per cent. did not.

(3) of 22 children with previous tonsillectomy, 17 or 77 per cent. were still subnormal, that is, suffering from varying degrees of debility, from one to eight years after tonsillectomy, despite no history of subsequent throat disorders.

The chemical findings in 70 of the children in whom the question of tonsillar disease was studied, are shown in Table 5.

TABLE 5.

COMPARISON OF CASES WITH AND WITHOUT TONSILLAR DISEASE.

Type	Blood sugar (mrgm. %)			$NaHCO_3$ (molar)			NaCl (grm. %)		
	No. of cases	Av.	Range	No. of cases	Av.	Range	No. of cases	Av.	Range
(b) Tonsils healthy	8	88	77—103	36	·0287	·0254—·0316	23	·607	·576—·638
(c) Tonsils unhealthy	7	99·7	89—116	34	·0286	·0245—·0315	22	·619	·588—·632

It will be seen that there is no significant difference between those with, and those without, tonsillar disease, as regards the chemical findings in the blood.

Table 6 shows the results of similar observations on the 22 children who had had a previous tonsillectomy. Here again, it will be seen that there is very little difference between subnormal children who have had a previous tonsillectomy, and subnormal children who have not, though it must be admitted that the plasma bicarbonate is a little higher in the former, than in the latter. It is interesting in this connection to recall the evidence regarding the results of tonsillectomy in such specific diseases as acute rheumatism and acute nephritis. It has been shown by many investigators, that this operation is of extremely doubtful value in both diseases, especially as regards recurrences, indicating that this operation does not generally lead to sufficient improvement in general health to protect against recurrence of the disease, once infection has occurred, although in both these diseases the tonsils themselves probably provide the primary portal of entry for the infection.

TABLE 6.

TONSILLECTOMIZED CASES.

Type	Blood sugar (mgrm. %)			$NaHCO_3$ (molar)			NaCl (grm. %)		
	No. of cases	Av.	Range	No. of cases	Av.	Range	No. of cases	Av.	Range
Normal	—	—	—	4	·0301	·0312 ·0295	5	·596	·608 ·587
Subnormal	3	101	109 92	18	·0279	·0303 ·0228	13	·610	·632 ·588
Total	3	101	109 92	22	·0290	·0312 ·0228	17	·603	·632 ·587

Paterson and Gray[9] showed that in a series of subnormal children, marked improvement in general health, and increase in weight, was found up to 6 months after tonsillectomy, a result which, at first sight, strongly suggests the importance of tonsillar disease as a cause of chronic debility in children. The Board of Education[10], however, found that if a series of subnormal children in whom the tonsils had previously been removed, were followed up for a considerably longer period after operation than six months, the initial improvement noted by Paterson and Gray, was not maintained. Further evidence relating to this problem has been reported by one of us (H.G.C.) elsewhere[11].

The balance of evidence would seem to suggest that tonsillar disease is not such an important factor in the production of chronic debility in children as is usually supposed, and that tonsillectomy is an operation which is often performed unnecessarily.

Relation of debility to cyclical vomiting.

In a paper already referred to above[6], it was pointed out that cyclical vomiting was a disorder met as frequently amongst the children of the hospital classes as amongst those of the well-to-do. This, again, is contrary to the usual teaching. It was also stated that cyclical vomiting occurred in about 30 per cent. of all elementary school children. The evidence for this was based on a history of the symptom-complex described above in 500 consecutive children between the ages of 6 to 12 years, attending hospital for minor disorders. In actual fact, cyclical vomiting, as judged by these standards, occurred in 45 per cent. of the series referred to, but in order to eliminate possibility of error arising from the necessity of asking leading questions in a number of instances, an allowance of 15 per cent. was made. The figure of 30 per cent., therefore, may be taken as a conservative estimate of the frequency of this disorder in the children of the hospital classes. E. C. Warner, working in the Children's Department at Guy's Hospital, informs us in a personal communication that, adopting the same criteria as we had done, he had found that 20 per cent. of a series of 300 children out-patients, between the ages of 3 to 12 years inclusive, suffered from major attacks of cyclical vomiting. As this disorder does not usually appear in its most typical form before six years of age, it is clear that, allowing for this, 30 per cent. is again a fair estimate of the frequency of cyclical vomiting in children of this social class.

In the present series of 108 children, a definite history of major attacks of cylical vomiting was obtained in 47 cases. Table 7 compares the biochemical findings in 16 children with a history of cyclical vomiting, who had not recently had an attack, and 14 subnormal children who gave no history of this disorder.

These results show that there is no biochemical difference, in respect of the observations made, between these two groups. Of the 39 normal children, 13 to 33 per cent., gave a history of cyclical vomiting, and of the 70 subnormal children, 35 or 50 per cent., suffered from this disorder. These figures, though not very large, suggest that cyclical vomiting is more often found in the unfit,

than in the fit child, and still further emphasizes the clinical fact, hitherto overlooked, of the frequency with which this condition is met in children of the poorer classes.

TABLE 7.

COMPARISON OF CASES SHOWING CYCLICAL VOMITING WITH SUBNORMAL CASES WITHOUT IT.

Type	Blood sugar (mgrm. %)			$NaHCO_3$ (molar)			NaCl (grm. %)		
	No. of cases	Av.	Range	No. of cases	Av.	Range	No. of cases	Av.	Range
C.V.	16	99·7	75—146	30	·0277	·0228—·0316	10	·610	·576—·638
G.	14	98·6	77—139	35	·0279	·0254—·0316	17	·601	·588—·623

It is a matter of common observation, referred to by all authorities on the subject, that cyclical vomiting is most apt to occur and to be most intractable, in the highly-strung, excitable child, and to this extent the biochemical results given in Table 7, may be regarded as typical of the nervous child; but as mentioned elsewhere[6], cyclical vomiting is a disorder which occurs perhaps in its most dramatic form in the child of nervous make-up, but it is by no means entirely confined to such. This is opposed to the view expressed by some that the acidosis and disturbances of carbohydrate and fat metabolism, with which it is said to be associated, constitute a biochemical picture peculiar to the nervous child.

It is at least apparent that cyclical vomiting is a disorder of very frequent occurrence amongst children of all social classes; that children who suffer from it do not differ in respect of the biochemical observations made herein, from those who do not, and that it is not the prerogative of the nervous child.

Observations on the treatment of debility in children.

About seven years ago, one of us (A.A.G.), when collecting cases of cyclical vomiting for investigation in the Children's Department at Guy's Hospital, observed that those children who had been given extract of malt, largely with the object of securing regular attendance at out-patients, ceased to have further attacks of the disorder. This at once suggested that sugar in some form was the needed antidote. This hypothesis was rendered more probable, by the fact that, in an undue proportion of these cases, it was noticed that sugar was being withheld, as far as possible, as the mother had been told that it was 'bad for the teeth,' 'brought the child out in spots,' or, 'was bad for children.' a doctrine which incidentally, is still held, and acted upon, even in some of the best public schools in the country. There is, of course, no adequate scientific evidence for any of these suggestions.

In order to test further the action of sugar in preventing cyclical vomiting, a group of 30 children suffering from this disorder was given extra sugar,

and kept under observation for from one to three years, and as a result, it was found that with very few exceptions, attacks of cyclical vomiting ceased to occur. It became apparent too, that not only was cyclical vomiting easily prevented by this simple and harmless measure, but that a large number of the other common symptoms of debility in children, such as headaches, recurrent abdominal pains, constipation, ' growing pains,' pallor, and undue fatiguability, were also cured, and prevented, and that the giving of extra sugar, in any form, quite frequently resulted in a rapid and marked improvement in the general health, spirits, and appearance of the child. In fact, without any other form of treatment, or alteration in the daily régime, a pale, dispirited, ailing child, would often be converted into a high spirited, healthy, and happy being. This form of treatment was later adopted by Dr. H. C. Cameron[13, 14], and he has confirmed the beneficial results obtained. As pointed out elsewhere[6], cyclical vomiting occurs in three main forms:—the classical cyclical, or periodic vomiting, cyclical diarrhœa, and cyclical pyrexia, each of which may occur singly or together. Quite early in the investigation, it was noticed that there was also a recurrent, or cyclical, ' respiratory ' group, the symptoms of which were indistinguishable from those of ordinary bronchial asthma. In view, however, of the well known difficulty of differentiating between some cases of asthma and recurrent bronchitis in young children, it was considered unwise to assert that asthma could be successfully treated by sugar, but it soon became obvious that these cases were, in fact, true examples of bronchial asthma, and that at least in some of them, sugar was an effective prophylactic. Although it has been shown that the most common and constant biochemical feature of debility in children is an ' acidosis,' it is a remarkable fact that, in our experience, the giving of alkalies by mouth does not in any way lead to improvement in the condition, or in any of its numerous and varied symptoms. As far as we have been able to ascertain, also, alkalies are quite without value in the treatment or prevention of cyclical vomiting and asthma in children. There is, however, one point in this connection, which should be borne in mind, before concluding that alkalies are without value in these cases, and that is, the question of increased liability to infection, where some degree of acidosis is present. Cameron[12] asserts that children suffering from acidosis are predisposed thereby to infection. We have long had this possibility in mind, and have endeavoured to obtain evidence upon the question, but at present, it can only be said that no conclusions have been reached. If the type of acidosis referred to here should be shown to predispose to infection, then it is possible that the use of alkalies in these cases may be justified.

Conclusions.

(1) The state of subnormal health in children, usually called ' debility,' is generally associated with some degree of ' acidosis,' defining this as a reduction of the plasma bicarbonate only.

(2) Debility in children is not commonly accompanied by hypoglycæmia, and the term ' glycopenia ' is inaccurate, and should not be used to describe these cases.

(3) The acidosis of debility is not generally due to an associated acetonæmia or ketosis.

(4) Ketosis in children is not generally associated with hypoglycæmia.

(5) Tonsillar disease is not the most common cause of debility in children, and tonsillectomy is an operation which is often performed unnecessarily.

(6) Cyclical vomiting is a disorder which occurs with great frequency amongst children of the so-called hospital classes, the incidence in this class being about 30 per cent. of all children between the ages of 6 to 12 years inclusive.

(7) The biochemical picture associated with disturbances of the carbohydrate and fat metabolism, acetonæmia, etc., is not more frequently found in 'nervous,' than in other subnormal children.

(8) There is no specific biochemical picture peculiar to the 'nervous' child.

(9) There is no evidence that alkalies are of value in the treatment of debility in children.

* * * * *

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